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DISEASES OF CHILDREN

A TEXT-BOOK FOR THE USE OF
STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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PREFACE TO THE THIRD EDITION

The present work, while based upon the previous editions of the author's *Diseases of Children*, has been entirely re-written. The bulk of the material is, therefore, new; this applies particularly to the chapters on infant feeding, diseases of the digestive tract, infantile diarrheas and constitutional diseases. It has also been necessary to add a number of new articles, for example—lethargic encephalitis, myatonia congenita, spasmophilia, the exudative diathesis and acidosis. On the other hand, some topics which were discussed in the former edition are omitted as they were of purely surgical interest. Illustrations have also been omitted in an effort to keep down the bulk of the book. The author feels that they are not essential to a work of this size and scope.

The prime objective of the writer has been to present to the profession a work which will answer the purposes of a textbook for students and a reference book for the busy practitioner. He has, therefore, endeavored to give not only a concise description of the diseases of children but also full instructions in the care and feeding of infants and children and in the treatment of the diseases under discussion.

The homeopathic treatment of the diseases of children is fully discussed and is based upon the author's many years of experience in this field. The indications for the selection of the homeopathic remedy as well as the dosage and method of administration are presented in such a manner that physicians

who have not had special training in homeopathic materia medica but who desire to use homeopathic treatment in their practice can get the information and help they are seeking. The chapters on Homeopathic Therapeutics and Homeopathic Prescribing in Diseases of Children have been especially written with this point in mind. The author desires to say, however, that he has felt free to discuss and present all methods of treatment which are generally accepted as possessing specific clinical value irrespective of their mode of action and wherever their use is advisable or is imperative, he has unequivocally recommended them.

Philadelphia, June, 1922.

PREFACE TO THE SECOND EDITION

Since the appearance of the first edition of this work a number of important discoveries have been made in the field of Pediatrics, and some significant changes have occurred in the views held at that time regarding the etiology and treatment of not a few of the commonest affections in childhood. Furthermore, it is but fair to say that the writer himself has felt the need for revising some of his views expressed in the former edition, for with riper years and larger experience he has learned the value of conservative methods, and has endeavored to replace the mere possibilities of therapeutics with clinical certainties.

The text has been entirely rewritten, and new matter has been added wherever it was found desirable to amplify any subject. The chapter upon Infant Feeding is practically new, and the aim has been to present in a concise and clear form the most acceptable and modern views upon this subject, which has of late years been made unnecessarily complicated. A chapter upon Diseases of the Ear, Nose and Throat has been added, and illustrations have been inserted wherever a picture or a diagram could be advantageously employed to elucidate the text.

I am again indebted to a number of my colleagues for valuable suggestions and friendly co-operation, which, to my mind, is necessary in any work covering so broad a field as that of Pediatrics. Dr. Chas. M. Thomas has kindly read the sections dealing with the diseases affecting the eyes, the ears,

the nose and the throat, and has made a few additions to the manuscript. To Dr. Wm. B. Van Lennep I am indebted for assistance in revising the articles upon appendicitis and intussusception, and also for suggestions concerning the treatment of other conditions, wherever this has presented a surgical aspect. Dr. W. D. Bayley has kindly offered some suggestions relative to Mental and Nervous Diseases.

For the excellent index I am indebted to Dr. Ernest A. Farrington, whose painstaking arrangement of the various subjects mentioned and discussed must of necessity add to the practical value of the book. I have also to thank the publishers for their liberality in preparing the many illustrations, and for numerous other courtesies.

I cannot refrain from expressing my appreciation of the kind reception which the first edition received at the hands of the profession and of the students of our colleges, and while my aim has been not to overstep the bounds of a Text-Book, I trust that the busy practitioner will find within these pages all the practical information which he may need.

February, 1906.

PREFACE TO THE FIRST EDITION

In presenting this work to the profession the author has aimed to make it a purely clinical one.

In the sections on treatment he has endeavored to give his own experience as much as possible, and has sought to exclude all doubtful symptoms and theoretical indications.

The section on Skin Diseases is from the pen of Dr. Leon T. Ashcraft, Lecturer on Venereal Diseases at the Hahnemann College.

In the section on Nervous Diseases, valuable suggestions have been made by Dr. Weston D. Bayley, Lecturer on Mental Diseases and Clinical Instructor in Nervous Diseases at the Hahnemann College.

Philadelphia, 1899.

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DISEASES OF CHILDREN.

CHAPTER I.

HYGIENE AND NURSING.

The New-born.—The first step in the care of the new-born infant, after respiration has been established, is the care of the eyes. They should be thoroughly cleansed with a warm, saturated solution of boric acid, after which a drop of a 2 per cent solution of nitrate of silver is dropped into each eye according to the method of Crede. By carrying out this prophylactic measure *ophthalmia neonatorum* can in most instances be prevented.

After the cord has been dusted with powdered boric acid and dressed in sterilized gauze the child should be wiped dry, the body anointed with sweet oil, especially when there is an abundance of vernix caseosa, then wrapped in a warm blanket and laid aside until it is convenient to give it the cleansing bath. Asepsis in the care of the cord is of the greatest importance since the majority of cases of septic infection in the new-born originate at this site. The early dropping of the cord is favored by the use of dry dressings; salves and ointments should not be used. The stump must be carefully dressed until healed.

Bathing.—The full bath should not be given until the cord has fallen off. This usually occurs at the end of the first week. The child should be bathed in a warm room, preferably before an open fireplace. The first bath must be a warm one, approximating the normal body temperature; in hardy children it

can gradually be reduced, so that a temperature of 95 degrees F. may be reached, by the end of the sixth month. It should be of short duration and the body dried by light rubbing with a soft towel. The bath is best given in the morning before the second nursing. In children who do not react well the full bath may have to be discontinued and a sponge bath used instead. The use of too much soap or hard rubbing is a mistake and may cause troublesome dryness or irritation of the skin.

Clothing.—The material for the underclothing should be soft and non-irritating. In the summer it may be of cotton and for winter part wool and cotton. The clothing should fit loosely and there should be no constricting bands and heavy seams. Buttons can often be advantageously replaced by tapes. The abdominal binder should not be worn after the third month unless the infant is delicate and underweight or has an umbilical hernia. In the winter the band with shoulder straps may be worn as an extra protection to the infant's abdomen.

The diapers should not be too bulky or applied too tightly. Stockinet is light and more absorbent than linen and less expensive. Stress should be laid upon the importance of promptly removing soiled diapers and carefully washing them and then rinsing in plain water before they are again applied.

The Mouth and Teeth.—In cleansing the baby's mouth great care should be exercised not to injure the delicate mucous membrane and thus invite infection. The use of a swab of cotton dipped in a 2 per cent boric acid solution is a safer procedure than inserting the covered finger into the mouth. In the case of thrush borax should be used in place of boric acid.

The care of the teeth has an important bearing on the child's general health. Indigestion, enlarged tonsils and cervical adenitis are often directly traceable to dental caries. Malocclusion also seriously affects the child's health. As in the case of adults, we should always examine the teeth for a focus of infection in all obscure ailments of possible infectious origin.

Sleep.—The healthy infant is a good sleeper, usually not

being awake more than an hour at a time and only waking for the purpose of nursing. After the sixth month it gradually becomes more wide awake during the day, requiring usually one or two naps, and about twelve hours sleep at night until it is a year old. From this time on until the fourth year it should sleep twelve hours at night and have a daily nap.

Children should be carefully trained in regular habits of sleep. The most common causes for disorders of sleep are improper training, indigestion, frequent nursing during the night and sometimes local disturbances such as too much clothing, phimosis and seat-worms.

The Bowels.—The infant can be taught to form the habit of having regular bowel movements by placing it on a chamber held on the nurse's lap and starting the movement with a suppository if necessary. This training can be begun as soon as the infant is able to sit up.

The infant should have a bowel movement once in twenty-four hours at least. If allowed to go over until the next day the movement may become hard and difficult to pass and the anus may be torn. This leads to the development of a fissure with painful defecation and consequent troublesome constipation.

Airing.—The nursery should be sunny and well-ventilated without being draughty. If the child is allowed to crawl the floor should be suitably covered. A pen is an excellent device for keeping the infant out of harm and giving it full opportunity to exercise and learn to walk.

Ventilating the nursery in winter is best accomplished by having the windows open in the adjoining room until the air has been perfectly purified, when the windows should be closed and the communicating door opened to allow a diffusion of the atmosphere from one room to the other. Once a day, however, when the infant is out of the nursery, it should be thoroughly aired.

As a rule, in the spring and fall an infant may be taken out into the fresh air at one month and even earlier during the

summer. During cold weather, however, an infant under three months should not be taken out of the house, and after that age only during the sunny hours of the day. The precautions necessary to be observed in taking a child out in its coach are that it be kept out of the wind, that it be sufficiently covered and that the sun does not shine directly into its eyes, but there is no valid objection to allowing a child to sleep in the open air in element weather providing the above precautions are taken.

Statistics show that infants require more airspace, proportionately, than adults, and that overcrowding is a prolific source of ill-health among children. This is especially the case in institutions and hospitals for children.

Exercise.—The infant gets its exercise in crying and in the non-volitional movements which it performs. Playing with toys and creeping and walking soon become more effective forms of exercise, however. Older children require exercise of a more strenuous kind, such as walks in the open air, games, etc. A cold sponge bath every morning aids greatly in the physical development of the child. Fatigue and over-exertion in all forms of sport and exercise are to be carefully guarded against.

Premature and Delicate Infants.—The period of viability in a premature babe cannot be arbitrarily stated, as the condition of the infant plays an important role as well as its age. The period of viability has been usually fixed at twenty-eight weeks, but premature infants of twenty-four weeks have been successfully raised.

The fetus at the eighth lunar month weighs $3\frac{1}{2}$ lbs. and measures sixteen inches in length. The skin is wrinkled and the body is covered with lanugo. At the ninth lunar month it weighs 5 lbs. and measures eighteen inches in length. At the tenth lunar month, or term, it weighs 7 lbs. and measures twenty inches in length. The main problems with which we are confronted in caring for the premature infant are to maintain body-heat and to supply suitable nourishment.

Infants weighing four pounds and over can frequently be

reared without the use of an incubator. The body should be enveloped in a thick layer of cotton and in place of diapers, pads of gauze should be used. The entire body is then wrapped in a blanket and hot water bags applied to the feet and sides of the body or the infant may be placed upon an electrically heated pad. Daily inunctions with warm oil may be given but no baths.

Breast-feeding is usually essential to the successful rearing of the premature infant. The milk should be drawn from the breast with a pump and collected in a sterile receptacle. It is then best administered to the child with a medicine-dropper, the milk being dropped well back into the pharynx, four drams of nourishment being given every 2 hours. If the infant cannot be made to take sufficient nourishment in this manner, *gavage* must be employed. If breast-milk cannot be obtained whey may be tried or a weak milk formula of one part boiled milk to two or three parts water and five per cent sugar of milk.

The Care and Nursing of Sick Children.—In all acute illnesses the child should be kept in bed. Infants may occasionally be held in the mother's lap if they are very fretful and especially in the case of bronchopneumonia or protracted intestinal disturbances a change in the position of the patient is desirable. Unnecessary handling of a sick infant must, of course, be avoided. The child should be kept as quiet as possible, both mentally and physically; in the case of a serious illness only one attendant should be with the child at a time.

Temperature of the Sick Room.—The sick room must be kept well ventilated and not too warm, from 60 to 68 degrees F. In the summer an electric fan may be used to keep the air in motion but it should not be directed against the patient. The cold air treatment of pneumonia is only applicable to the lobar variety. Even in these cases it should not be carried to extremes.

Clothing.—In summer a light night shirt is all that is required; in cool weather the patient may also have a light undershirt to wear especially if the windows must be kept wide

open. Cotton-jackets, spice-bags and all forms of plasters are obsolete.

Heat.—Heat is perhaps the most useful of all non-medicinal therapeutic measures and has a wide field of applicability. In painful inflammatory conditions it acts promptly by relieving tension and hastening resorption. The old fashioned poultice is rapidly being replaced by hot fomentations. Dry heat is most conveniently applied by means of hot-water bags and heated flannel. It must be remembered that the child's skin is more sensitive and more readily burned than an adult's, for which reason proper precautions must always be taken.

Baths.—By means of the bath we are able to apply heat or cold most rapidly to the entire body. Hot baths are often useful in collapse and asphyxia neonatorum; by adding a tablespoonful of powdered *mustard* to the warm bath we have an excellent means of relieving serious congestion of internal organs, through its derivative effect. The *bran bath* is most useful in cases of eczema or other excoriating conditions of the skin.

The *tepid sponge-bath* must be used once or twice daily in all illnesses regardless of the presence of a rash or a chest condition. In case of high fever a *cold sponge-bath* may be given every two or three hours. The full cold bath is rarely used in children.

Packs.—Packs are highly efficient antipyretics and diaphoretics. The *cold pack* is applied by wrapping the child in a sheet wrung out of cold water, the sheet being surrounded by a dry blanket. When used to reduce fever it can be reapplied hourly. In pneumonia the pack is often restricted to the chest.

The *hot pack* is most useful in nephritis and uremia. A light blanket is wrung out of hot water and applied as above.

The *hot mustard pack* is prepared by adding a little ground mustard to the hot water; it is employed in convulsions, congestion of the lungs and of the brain; also to bring out tardy eruptions. While in the pack the head should be sponged with cold water or water and alcohol.

Inhalation.—The inhalation of steam is very beneficial in

respiratory ailments, especially in the case of croup. In the absence of a specially-constructed "croup-kettle," an ordinary tea-kettle heated by an alcohol stove or better, by an electric stove, may be used, the steam being directed under a sheet overhanging the child in the fashion of a tent.

Lavage.—The apparatus for carrying out lavage in children consists of a soft rubber catheter, connected with a piece of rubber tubing two feet long by means of a piece of glass tubing, and a medium sized glass funnel attached to the free end of the rubber tube. This apparatus may also be used for gavage. For the new-born infant a number 14 French soft rubber catheter should be used; in older infants a number 16 French scale is the proper size. In the new-born the catheter reaches the fundus, when introduced to the length of eight inches; in an infant of three months it must be inserted nine inches and in older infants from ten to twelve inches. I am in the habit of enlarging the eye of the catheter to facilitate the passage of mucus and curds. The passage of the tube into the infant's stomach is, as a rule, unaccompanied by any depression and causes only slight discomfort. Lavage is useful in all cases of persistent vomiting and is a necessary diagnostic procedure in certain gastric conditions, notably pyloric stenosis.

Lavage is performed as follows: The infant is held upright and seated on the nurse's lap, and covered with a towel to prevent soiling the clothing. The catheter is then inserted in the pharynx with the right hand, its tip following the index finger of the left hand which presses down the base of the tongue. Wetting the catheter with plain water is sufficient as a rule; if there be abnormal dryness of the mucous membrane, there is no objection to the use of olive oil. The child may make efforts at deglutition as soon as the catheter reaches the pharynx, in which case it glides down easily into the esophagus. More frequently, however, it gags, interfering with the operation. If we wait for a few seconds until the child draws in its breath, a gentle push will readily cause the catheter to enter the esoph-

agus. The catheter from now on reaches the stomach without difficulty. As soon as the catheter has entered the stomach the funnel should be lowered and the gastric contents permitted to flow out. Warm water or a weak solution of bicarbonate of soda are then poured into the funnel, a few ounces at a time, and allowed to run into the stomach and out again, until the water returns clear.

Irrigation of the Colon and Enemata.—For the relief of simple constipation and for emptying the lower bowel the *enema* is all that is required. This is best administered by means of a bulb syringe with the child lying upon its back. Soap-suds give the best results with the low enema.

When irrigation of the colon is to be carried out the child is placed on its left side upon a rubber sheet covered with a cloth, the hips being slightly elevated. A soft rubber catheter, number 18 or 20 French scale, is attached to the nozzle of a fountain syringe, lubricated and carefully inserted into the rectum. The water is then allowed to flow in slowly, stopping the stream as the child makes efforts at expulsion. The catheter is gradually inserted further and further into the rectum as the latter is distended by the water, until it has been introduced from six to eight inches. The fluid will usually reach the colon without difficulty, but cannot pass into the ileum, although it is claimed that if the colon is not distended and the water allowed to flow in slowly, a closure of the ileo-cecal valve fails to take place.

From six to eight ounces of warm, normal salt solution should be run into the bowel at a time and then permitted to flow out again through the catheter. After the process has been repeated several times the water usually returns clear and the operation may then be discontinued.

Irrigation of the colon is useful in diarrheal cases, especially if the stools are offensive or contain an abundance of mucus or curds. The same technique is employed when distension of the bowels with water is used in attempting to overcome an intussusception. *Enteroclysis* is a safe and efficient means of averting

circulatory failure in acute infectious conditions, and also for the purpose of aiding the elimination of toxins. This may be carried out according to the technique of Murphy, namely the "Murphy Drip."

CHAPTER II.

METHODS OF CLINICAL EXAMINATION.

The Period of Infancy and Childhood.—Infancy is the early period of childhood, the state of an infant in arms, or the first two years of life. Clinically we divide it into the period of the *newborn*, the *nursing period* and the *teething period* which terminates with the completion of dentition, namely, the end of the second year.

Childhood begins with the termination of the teething period and continues to the time of *puberty*. Certain diseases, not encountered in infancy now make their appearance, some of which are distinctive of childhood and may therefore properly be spoken of as “diseases of children.” The child is, however, also susceptible to many of the diseases of the adult, but its immature anatomical and physiological development so modifies their clinical course that they are quite different from the same disease as observed in the adult.

Mortality.—In recent years the death rate in early life has been markedly reduced through better sanitation and through the education of the public in the care of infants. In spite of preventive measures, however, a large number, approximately 10 per cent, of all infants die in the early weeks of infancy from congenital debility. Many of these no doubt could be saved were the mothers able to nurse them. The total number of deaths during the entire period of infancy is still 25 per cent.

Among the diseases responsible for this high death rate acute gastroenteritis heads the list furnishing about 30 per cent of all deaths. The acute gastrointestinal diseases of infancy occur almost exclusively during the summer. They are, to a great extent, preventable and therefore the outlook for a still greater reduction in infant mortality in the near future is hopeful.

Congenital debility, prematurity and marasmus contribute

25 per cent to the death rate. Acute respiratory diseases, which occur most frequently during the winter months are responsible for about 20 per cent of the deaths.

The History.—The first step in case taking is the interrogation of the patient, or in the case of an infant, the parent, for the purpose of ascertaining a history of the patient's ailment and of his state of health prior to the present illness. The family history must also be taken into account because of the important role played by heredity in many diseases.

The history should be kept in writing and accurate subsequent observations of the case added thereto, thus making a complete *record* of each patient under the physician's care.

The following data should be elicited in the order named and recorded in conjunction with the *physical findings* in the case:

Name of Patient,
Age,
Sex,
Family History,
Personal History,
Previous Illnesses,
Present Illness,
Physical Examination,
Laboratory Findings.

The *age* and *sex* are important to note as they will either suggest or exclude certain diseases. For example, during infancy, a bowel disturbance is much more likely to be ileocolitis than typhoid fever. Convulsions in infancy are more frequently functional and of less serious significance than in older children in whom they should suggest the probability of epilepsy. Again, sex may have an etiological bearing upon the case, certain conditions being more frequently encountered in one sex than in another.

The Family History should be inquired into for the purpose of determining the possibility of predisposition to or actual exposure to tuberculosis. A history of syphilis in the parents

can at times be obtained by direct interrogation. If this is denied, the history of repeated miscarriages and of previous still-born infants or of a previous infant dying with presumptive syphilitic manifestations will suggest the probability of this disease. Rheumatism, gout, diabetes and the physical vigor as well as the age of the parents should be inquired into. A neuropathic family history is frequently present in cases of nervous and mental diseases. The number of other children and their state of health should also be ascertained.

The personal history begins with the state of the mother's health during pregnancy. The influence of pre-natal impressions is doubtful; however, serious illness of the mother during pregnancy may affect the fetus to a marked extent.

The mode of birth is important as protracted labor or instrumental delivery may be the cause of a brain injury. The condition of the child at birth, whether asphyxiated and occurrence of cyanosis or convulsions at this time, are important data.

The weight at birth should be recorded and from it we can judge whether the infant's present weight is within normal limits. The mode of feeding, both past and present, must be known because of the importance of diet in the etiology of the nutritional disturbances of infancy.

The age at which the infant first sat up, crept, and walked and the time of the eruption of the teeth and closure of the anterior fontanel are indications of the infant's physical development. Its mental development can be judged from the age at which it began to play with toys, recognize mother or nurse and talk.

A list of the *previous infectious diseases* from which the child has suffered should be made. Since some of these will give immunity against a second attack while others cause increased susceptibility to a recurrence, this information may be of great diagnostic help. Again, certain of the infectious and contagious diseases leave sequelæ or they predispose to other infections. We should also determine whether the child has

shown a predisposition to certain ailments such as tonsillitis, rheumatic manifestations, bronchitis, gastrointestinal disturbances, nervous disturbances.

The child's environment must be inquired into as this may prove to be the explanation for the child's condition or give us the source of the infection in the case of an attack of malaria, typhoid fever, etc. Tuberculosis is also contracted by direct exposure or from "house infection." During epidemics the early recognition of such diseases as scarlet fever and measles is materially facilitated by the knowledge that the child has been exposed to either one of these diseases.

The nature of the *present illness* should now be recorded. The mode of onset, whether abrupt or with prodromata, the duration of the illness; the presence of fever—its character, whether uniformly high, remitting or intermitting. If other symptoms are present they should be carefully noted. Onset with vomiting, convulsions or sore throat are important data. The ratio of pulse, respiration and temperature should be considered. Pain can be suspected without interrogating the little patient; we can usually locate it by careful palpation or suspect its location from the child's actions.

Physical Examination.—The first step in physical examination is *inspection*. In order to inspect a patient properly he should be entirely stripped. The color, general state of nutrition, mental condition, attitude, facial expression and the presence of gross abnormalities such as a skin rash or a physical deformity can be taken in at a glance.

The healthy infant is well nourished and plump, there being an abundance of subcutaneous fat. The skin is pink and becomes red when the infant cries. Pinching the skin leaves no folds because of the vascular fullness of the integument (turgor). The babe is active, observing its surroundings with interest and the extremities are in motion. The temperature does not vary from the normal during different periods of the day (infantile monothermia).

The head of the infant under one year is slightly larger in circumference than the chest and abdomen, the latter being about equal. Variations from this standard should be noted; also general emaciation; pallor; cutaneous eruptions and cyanosis. Jaundice is frequently encountered in the new-born. Miliaria and sudamina are common in rickets. The *tache cerebrale* is a vasomotor disturbance encountered in meningitis. When the skin is irritated by sharply drawing the finger-nail across it a red streak will be left which persists for some time.

If there be a deformity of the *spine*, we must determine whether it be due to Pott's disease, rickets, a unilateral pleural effusion, old pleuritic adhesions, or muscular weakness. The child should be laid flat upon its stomach and the body then lifted from the table by the feet. If rachitic, the deformity is at once reduced, but the kyphosis of Pott's disease is irreducible under all methods of manipulation. Retraction of the chest from pleuritic adhesions produces scoliosis, and in these cases we can get the history of a former empyema.

The head presents many diagnostic features. In rickets it is large and square; in hydrocephalus large but rounded, the fontanels are widely open and the eye-balls displaced downwards. In rickets there are often parchment-like areas representing a thinning out of the bony elements, known as *craniotabes*.

The facial expression often points to the seat of trouble; for instance, the knitting of the brows in headache, which, when associated with strabismus, is a strong presumptive sign of meningitis; the fan-like motion of the *alæ nasi* in respiratory troubles, and the pinched expression of the face in abdominal disease. Roughly speaking, it can be said that the upper part of the face represents cerebral, the mid-portion respiratory, and the lower portion abdominal disturbances. Often one cheek will present a circumscribed redness, which is said to correspond to the side affected in pneumonia. This, however, is due to the cheeks having been lain upon and indicates vasomotor paresis.

The chest may present deformities, peculiarities of the ribs, deviations from the normal respiratory movements, abnormal movements and various skin eruptions. In the early stages of pleurisy the painful side becomes fixed and may produce a slight scoliosis. As the effusion is poured out the side bulges. In chronic pleurisy with adhesions the side becomes permanently retracted.

In rickets the sternum is prominent from lateral compression of the costal cartilages (pectus carinatum) and the pathognomonic beading of the ribs, the "rickety rosary," is often present. In phthisis that portion of the chest over the consolidated lobe is flattened and does not move in the same degree as the unaffected side, the clavicle stands out prominently and there is often marked retraction of the ribs (flattening) in that region. The long, narrow chest with acute epigastric angle is found in asthenic individuals who are predisposed to phthisis and enteroptosis.

In emphysema the chest assumes a rounded fullness, slight motion only being perceptible during respiration. After pericarditis with adhesions the intercostal space is often seen to retract distinctly during the heart's systole. In pericarditis we may also note *Broadbent's sign*, i.e., systolic retraction of the lower ribs posteriorly on the left side.

The *limbs* and *joints* must be examined for evidences of rheumatism or tubercular arthritis; the deformities of rickets and poliomyelitis anterior; the bone affections of syphilis and tuberculosis; the marked tenderness and subperiosteal hemorrhages of scurvy should not be confused with arthritis.

The reflexes. Among the superficial reflexes the plantar is of especial importance. Under normal conditions a flexor response is obtained, but in lesions of the pyramidal system hyperextension of the great toe occurs. This is known as *Babinski's sign*. In infants up to the age of learning to walk the response may be similar to the Babinski phenomenon. The great toe is drawn back; the toes are extended and spread out

and the foot is everted. The Babinski sign is more deliberate, however, and there is but a small amount of movement at the ankle.

The knee-jerk is exaggerated in lesions of the upper motor neurons or irritation of the lower neurons. Diminished or abolished knee-jerks indicate a lesion in the lower motor neurons. In children it is best obtained in the dorsal position with the foot resting on the palm of the left hand, striking the tendon with a percussion hammer held in the right hand.

Ankle clonus indicates disease of the spinal cord, from the first to the third sacral segments.

The position assumed by the child during sleep and waking is important to note. We see the child burying its head in the pillow when there is photophobia; lying on the back with limbs drawn up in abdominal inflammations; persistently lying upon the side in acute pleurisy; the head drawn back and the neck rigid in meningitis; unable to lie in the prone position in the dyspnea of capillary bronchitis and heart disease; impossibility of extending the leg upon the thigh when in the sitting position owing to the contraction of the flexor muscles, which disappears when the dorsal decubitus is assumed (*Kernig's sign* in meningitis); sleeping or comatose; crying out in sleep and gritting the teeth. During natural sleep the child assumes a position indicating complete relaxation, the respiration is of the abdominal type. Infants sleep with the arms drawn up and flexed, probably the position maintained during intra-uterine life.

The character of the *cry* is often a help in diagnosis. The shrill, piercing cry of meningitis is pathognomonic. The hoarse cry of the new-born infant suggests syphilis. In otitis the cry is often continuous in spite of all that is done to humor the child. The recognition of the cry of hunger, pain and temper is more readily attained by observation than from reading. The natural cry is a loud, strong vocal effort accompanied by reddening of the face and does not last more than a few minutes. Abnormal cries are, as a rule, weaker in character and more

persistent. The cry of pain may be strong, but it is accompanied by evidences of sufferings and distress, such as facial contortions tears, drawing up of the legs or bringing the hand to the affected part. The cry of hunger is a continuous, fretful cry, ceasing when food is offered.

The inspection of the *throat* is left until the last because it usually causes the child to cry and so interferes with further examination of the case. The child must be firmly held and it is usually necessary to gag it in order to get a satisfactory view of the throat. Infants may be rolled in a sheet with the arms extended at the sides in order to prevent struggling and older children can be taken on the nurse's lap, the legs held between the nurse's knees and the arms and head controlled by the nurse passing her arms under the child's axillæ from behind and then over the back of the head. However, children can be taught early to have the throat examined and if they are not unnecessarily gagged will offer little objection.

In contagious diseases we should be prepared for the sudden cough which is likely to occur and spurt infectious material into the examiner's face. Often it is only necessary to allow the child to cry during which act a satisfactory view of the throat and mouth may be obtained.

Palpation.—The sense of touch, properly trained, will give much valuable information in the study of sick children. The first thing that strikes our attention as we touch the child's body is the temperature. Fever, however, cannot be accurately estimated by palpation alone. Dryness or moisture of the skin as well as roughness should be noted. Palpation of the head determines the state of the anterior fontanel and the sutures; the presence of cranio-tabes; mastoid tenderness.

From the head we descend to the *chest*, taking also the *neck*, where we often find scrofulous enlargements of the cervical glands. In an examination of the chest palpation is usually the first step, and if the child is crying we can determine the vocal fremitus. The child should be held by the mother in

such a manner that it rests on one of her shoulders and presents its back to the physician. The hand is then placed on the back in order to determine the vocal fremitus; the rattling of mucus in the bronchi is distinctly transmitted to the hands, in bronchitis. The hands can then be placed on the sides of the chest and the respiratory movements of both sides compared. The left hand will now seek the cardiac area, by which means hypertrophy or a thrill can often be detected.

The abdomen is most satisfactorily palpated while the child is asleep, the warmed hand being gently introduced under the bed-covering. Distension or retraction of the abdominal wall was noted while inspecting. The trained palpating hand will recognize enlargement of the liver and spleen; the presence of enlarged mesenteric glands; impacted feces, etc. The abdominal muscles lose their normal tone in disease or they become rigidly contracted in abdominal inflammations.

Abdominal pain may be a "referred pain" from the chest, but localized tenderness is of great diagnostic importance. Tenderness over McBurney's point and rigidity of the right rectus is pathognomonic of appendicitis. Gurgling in the right iliac fossa together with tenderness is strong presumptive evidence of typhoid fever, but this is not a pathognomonic sign.

The bladder may be felt in the hypogastrium when distended. A *rectal examination* should be made as a supplement to the abdominal examination in all doubtful cases.

Percussion.—Percussion of the head is employed in eliciting Macewen's sign. The skull is percussed directly with the finger and in the case of distension of the lateral ventricles with fluid a hollow note is heard. It is an early sign of meningitis.

In percussing the *chest* of a young child we must bear in mind the lesser dimensions of the thorax and the greater elasticity of the chest walls. This anatomical fact makes it more difficult to outline the heart boundaries than in older children. The thinness of the chest wall and the relatively large size of the bronchi gives the percussion note a hyperresonant character. Therefore light percussion should be used.

For percussion of the front of the chest the child should lie upon its back. For percussing the back, it should be held over the nurse's left shoulder with its arms about the nurse's neck. This is also the best position for auscultation.

Percussion of the upper dorsal spines and the interscapular region for eliciting the sign of d'Espine is most satisfactorily performed with the child in the sitting posture and with the head reflexed. In this position the region of the bronchial glands is also best auscultated.

In the percussion of a superficial organ (thymus) or the superficial area of dulness of the heart (the "absolute dulness") the best results are obtained by using the middle finger of the left hand for a pleximeter and striking quick, gentle taps with the middle finger of the right hand. When striving to elicit deep dulness in order to outline a deep-seated organ like the spleen or to determine the deep ("relative") dulness of the heart or liver, the finger should be pressed deeply into the intercostal spaces and the percussion strokes dealt more strongly, avoiding loud percussion, however, which drowns out the finer shades of distinction between the notes and practically abolishes all border lines. In percussing, the examiner's finger also experiences the sense of *resistance* which is of especial aid in the detection of fluid in the chest.

In percussing the chest, normally, the percussion note gradually increases in intensity both anteriorly and posteriorly as we descend and then gradually diminishes as the lower border of the thorax is reached. The increase in intensity in percussing downwards results from greater thinness of the thoracic wall—the pectoral muscles and the scapula and its muscles padding the upper part of the thorax considerably—and the flatter configuration of the chest at its mid-portion. As we descend we impinge upon the deep dulness of the liver and spleen posteriorly and the liver and heart anteriorly.

The lower border of the lungs in the dorsal position is identical in children and adults, and not higher as Weil claimed

(Sahli). The following points reach the extreme lower border of the lungs: Right mammary line, upper border of the sixth rib; left mid-axillary line, upper border of ninth rib; posteriorly, on either side of the spine, eleventh dorsal spine.

The percussion note over the *sternum* is more intense than in the adult owing to the elasticity of the thorax. A slight shade of difference naturally exists and is apparent in percussion from an adjacent region of the thorax toward the sternum and over it, but it is not more pronounced than is the difference existing in the note over a rib, and in an intercostal space. Percussion of the sternum in children, therefore, gives more positive results than in the adult. In percussing from above downwards the upper boundary of the deep cardiac dulness may be traced by means of light percussion. The presence of the *thymus gland* may also be demonstrated in the upper sternal region in young children, when hypertrophied.

Auscultation.—The position for auscultation is the same as recommended for percussion. Auscultation is most satisfactorily performed when the child is quiet but during crying we can estimate the vocal resonance and also hear rales at the end of inspiration. Crying is purely an expiratory act and therefore does not interfere with inspiratory sounds.

Older children may be engaged in conversation when we wish to study the voice sounds. A binaural stethoscope with a small bell or chest-piece, is the instrument to be recommended.

The heart may be auscultated posteriorly almost as well as anteriorly in infants and the murmurs of congenital heart disease are often better heard between the scapulæ than over the cardiac area in front. The heart should always be examined as a matter of routine and before the infant begins to cry as it cannot be auscultated during crying.

Normally the first sound at the apex is the loudest sound of the heart. Next in intensity is the pulmonary second and lastly the aortic second sound. The rhythm is trochaic (Hochsinger). The pulmonary second sound is normally louder than

the aortic second up to the tenth year. The first sound of the heart develops its booming, muscular quality during the second year. Prior to that time the heart sounds are of the embryonic type.

Auscultation is seldom of use in abdominal conditions in children, excepting to determine the absence of intestinal movements (peritonitis).

The respiration of the child is of the *puerile* type, characterized by harsh respirations, somewhat bronchial in character. In infants, owing to the slight movements of the chest wall and the purely abdominal type of breathing, the respiratory sounds are feeble.

As the right bronchus is of larger calibre than the left, the respiratory note is more intense on the right side. Broncho-vesicular breathing may normally be heard to the right of the spine in the scapular region.

Occasionally during deep inspiration, especially during crying, sub-crepitant rales may be heard at the apices (supra-clavicular region) and at the bases posteriorly. In pneumonic conditions we must be careful not to confuse harsh, rasping subcrepitant rales, characteristic in children, with pleuritic friction sounds.

Pulse, Temperature, Respiration.—The pulse is normally rapid in children, gradually decreasing in frequency during childhood, attaining the average rate of 76 in males and 80 in females by the time of puberty. In young children the rhythm is variable and irregular, being influenced by the respirations (sinus irregularity). The pulse-rate is not a safe criterion of fever, which can only be accurately determined by means of the clinical thermometer.

During the first weeks of life the pulse-rate varies between 125 and 135 beats per minute. From the sixth to the twelfth month it is usually 105 to 115 and is more susceptible to crying, etc. From the second to the sixth year it may be said to vary within 90 to 105 beats; seventh to tenth year 80 to 90 beats, after which it gradually attains the average adult standard.

One of the most satisfactory results to be obtained in studying the pulse is when we compare it with the temperature and respiratory ratio. Thus in the beginning of typhoid fever the temperature may have risen several degrees above normal while the pulse rate is still unaffected. Later it may rise entirely out of proportion to the temperature. The pulse does not, therefore, rise in uniform ratio with the rise of temperature in all cases, although, as a rule, *one degree of fever-heat is usually accompanied by an increase of eight pulse-beats.*

The number of respirations per minute does not correspond so closely to the temperature as the frequency of the pulse. In pneumonia the rate of respiration is entirely out of proportion to the fever and the pulse, and greatly quickened respirations should at once lead us to examine the chest.

The temperature is best taken by inserting a clinical thermometer, lubricated with vaseline, into the rectum. It is usually a trifle higher than in the mouth, but it is much more satisfactorily taken here, and far more accurately than in the axilla or groin. Diurnal variations in the temperature are of the same significance in children as in adults.

The Urine.—Routine examination of the urine in infancy is just as important as in older children and should not be neglected because of the slight trouble necessary to obtain a specimen. Many cases of albuminuria and pyelitis are overlooked by a failure to make urinalyses especially in cases of fever of obscure origin.

Specimens of urine can readily be secured in male infants by attaching a test-tube to the penis and leaving it there until the child has urinated. In the case of female children a small cup may be held in place over the vulva until a specimen is obtained. In many instances it is only necessary to seat the infant on a chamber and it will void urine voluntarily.

The daily quantity of the urine increases gradually from an ounce at birth to six to ten ounces by the end of the second week. The amount is relatively large during early infancy, in-

creasing from six to twelve ounces at one month to sixteen ounces at six months of age. By the second year it may reach twenty ounces, and by the eighth year one quart. The specific gravity is relatively low during infancy, the percentage of solids also being low, but the amount of urine passed is greater in comparison with the body-weight than in adults. The new-born voids a concentrated, highly colored urine leaving a pinkish stain of urates and uric acid upon the diaper. Micturition is characteristically frequent during infancy and the urine is passed unconsciously by the infant. At the end of the second year the child becomes conscious of the desire to urinate and it should learn to control urination at this time.

CHAPTER III.

THERAPEUTICS.

The treatment of disease according to the homeopathic method is based upon the law of similars enunciated by Hahnemann in his *Organon of the Art of Healing* which he expressed by the formula "*similia similibus curentur.*" There has been much arguing pro and con as to whether this formula represents a law of cure or whether it is only a rule of practice. This question is of minor importance, however, and is not essential to an understanding of the principles of homeopathic practice. The latter is based entirely upon the homeopathic materia medica which has been developed from studies of the effect of drugs upon healthy individuals, the symptoms induced by the ingestion of the various drugs being carefully noted and classified and thus a more or less complete symptomatology of each drug obtained. This method of study was designated by Hahnemann as "drug proving." The homeopathic materia medica also includes in its symptomatology the pathological effects of drugs observed in animal experiments and in human beings who have been poisoned by such drugs whenever these data are available. In this manner the pathological action of drugs has been learned and the homeopathic relationship of certain drugs to certain diseases has been established. Again, some of the symptoms which have crept into the materia medica are entirely empirical; their therapeutic usefulness having been established by clinical experience. No claim is made that such symptoms always indicate homeopathic drug action and we are fully aware of the fact that the homeopathic materia medica needs a re-proving of many of its drugs. However, these empirical, or "clinical" symptoms should be accepted for what they are worth and not used as an argument against the homeopathic method.

The isopathic principle has become a most important factor in modern therapeutics; this is especially seen in the strides made by vaccine therapy in recent years. The cure of anaphylactic conditions such as hay-fever and asthma by the use of foreign proteins for the purpose of de-sensitization is another example of isopathy. This principle has also been applied to the cure of poison ivy dermatitis with the tincture of rhus toxicodendron by Schamberg (*Jour. Amer. Med. Asso.*, Oct. 18, 1919). While these isopathic methods are, strictly speaking, not homeopathic, still they embody the same idea of the use of a single remedial agent administered in small doses for the purpose of bringing about a systemic curative reaction.

In the treatment of the sick all accepted therapeutic methods have their place. Purely physiological effects such as are obtained from the administration of strychnia, digitalis and the bromides are needed in certain instances. Palliatives are frequently called for and are necessary adjuvants to the armamentarium of the physician. Specifics also occupy their proper place in medicine, notably quinine in malaria and mercury in syphilis. The former is an example of chemotherapy; the latter is a striking example of *similia similibus curentur*. The symptoms induced by small doses of mercury in individuals with an idiosyncrasy to this drug and the pathological manifestations of chronic mercurial poisoning present a similarity to many of the symptoms of syphilis which cannot be explained as a mere coincidence.

Dosage.—The history of the homeopathic dose is given by Richard Hughes in his *Manual of Therapeutics* as follows, "When Hahnemann first began to prescribe medicines according to the rule 'similia similibus' he gave them in the usual quantities. It is not surprising that his patient's symptoms, even though ultimately removed, were often in the first instance severely aggravated. So Hahnemann reduced his dose accordingly. At what stage of this reduction he found that fractional quantities of a smallness hitherto undreamt of exercised a potent

influence, I cannot say. But once satisfied of the power of infinitessimals, he adopted them with enthusiasm as a part of the new system of medicine he was inaugurating."

For a long time Hahnemann's opponents looked upon his infinitesimal doses as scientifically undemonstrable and therefore unworthy of any serious consideration. We have learned in recent years, however, that a number of substances exert their specific effect in doses which are practically inconceivable and too small for demonstration by ordinary chemical or physical tests. The internal secretions and vitamins belong notably to this group. Granting, however, the possibility of the action of drugs in infinitesimal doses, still there is no necessity for the employment of infinitessimals in order to practice homeopathy. The dose in which the remedy is used is not the principle upon which homeopathy is based. The full physiological dose, however, cannot be employed for the purpose of obtaining a homeopathic curative effect. Hahnemann's original intent in recommending smaller doses than the usual physiological one was to avoid the production of medicinal aggravation of the symptoms present. Thus, while small doses of ipecac will relieve certain forms of vomiting, a full dose would aggravate the same.

To the beginner in homeopathy and to those not in sympathy with the theory of drug attenuation small doses of the tincture or the lower dilutions and the lower triturations are recommended. Let the dose just fall short of producing a medicinal aggravation and if the remedy is homeopathically indicated a curative effect will follow. Accordingly, the liquid remedies, excepting the very poisonous ones, may be administered in doses of one or two drops of the first or second decimal dilution and repeated every two hours or more frequently if necessary in acute conditions. In young infants the use of the third decimal dilution is preferable. When making use of insoluble substances such as the carbonate or phosphate of lime (*calcareo carbonica* and *calcareo phosphorica*), the phosphate of iron

(*ferrum phosphoricum*), the red iodide of mercury (*mercurius iodatus ruber*), etc., the third decimal trituration should be employed. Some of these insoluble substances notably the mercurial preparations are, however, active even in their crude state. The interesting experiments conducted by Dr. Percy Wilde and published in the Journal of the British Homeopathic Medical Society, Jan., 1902 demonstrate that the process of trituration induces changes in the physical property of the substance thus treated, converting apparently inert substances into a state in which they can enter into chemical combination with the cells of the human economy.

PREScribing IN DISEASES OF CHILDREN.

Prescribing homeopathic remedies for children presents certain apparent difficulties which are not encountered in prescribing for adults. Infants and young children are not able to tell us concerning their pains, discomforts and sensations and when they are old enough to answer questions the answers are frequently misleading. Pains are, as a rule, incorrectly located and their true location must be ascertained by our clinical skill. Such modalities as aggravation or amelioration from rest or motion; from hot or cold applications; from pressure and from lying upon the affected side; and such symptoms as nausea, thirst, photophobia, sore throat, tenesmus, etc., cannot be elicited by questioning the little patient. However, they can all be recognized by careful observation, and the history, inspection and the physical examination of the patient will give more accurate information than interrogation.

The selection of the homeopathic remedy should be made by a process similar to that employed in arriving at a diagnosis. First of all we should endeavor to determine the seat of the trouble and the nature of the pathological process which is responsible for the symptoms present. In acute illnesses there may be an infection with predominance of local or general manifestations. Fever will be present in all of these conditions.

When upper respiratory symptoms predominate *aconite* and *gelsemium* are indicated. They must be differentiated by the appearance of the child, in the case of *gelsemium* it is heavy and listless, there is general aching and lassitude as in grippe and the fever is not high. In the case of *aconite* the fever is high and is ushered in with a chill; the patient is anxious and restless and tosses about from side to side. The feel of the skin may be deceptive as to the degree of fever present. In cases in which *aconite* is indicated the body surfaces may feel cool especially during the chilly stage while the rectal temperature may register 103° to 104° ; the *belladonna* case, on the other hand, owing to the dilatation of the cutaneous vessels, presents a hot body surface and the child may appear to have a very high fever when in fact the rectal temperature will register lower than in the former instance. *Gelsemium* is indicated in simple coryza and grippe cases while *aconite* is more suited to the beginning of bronchial and pulmonary inflammations. When throat symptoms dominate the clinical picture *belladonna* will suggest itself rather than *aconite*; if joint symptoms are present we will incline to *bryonia* rather than to *aconite* or *belladonna*. Fever alone is therefore not sufficient clinical basis for a prescription and that is why the homeopath gives *aconite* in one case, *belladonna* in another and *gelsemium* in still another type of case.

The appearance and behavior of the child is of great help in prescribing. It may be pale or flushed; the skin hot and dry or moist; it may be listless and apathetic or restless and irritable. It may lie with its face buried in the pillow, or turning from side to side, or carefully maintaining a fixed position on one particular side. It may lie flat on its back and make no effort to move. These are all symptoms which suggest certain remedies just as they suggest certain clinical conditions.

Gastrointestinal symptoms which are purely due to dietetic errors are of little significance from the standpoint of the prescriber because the correction of the diet is all that is neces-

sary. Such symptoms as the vomiting of tough curds or the passage of curds in the stools; colic; constipation and simple diarrhea promptly disappear when the milk is properly modified and given in the right amount and at the right intervals of feeding. When, however, the dietetic factor has been active long enough to bring about an inflammatory reaction in the mucous membrane of the gastrointestinal tract or when infection is added, then a condition arises in which it not only becomes necessary to remove the cause as far as we can by a strict regulation of the infant's food but we must also prescribe for the symptoms which have associated themselves with the dyspepsia. The same holds good in the case of the dyspeptic and nutritional disturbances which result from prolonged improper feeding. The food intolerance; the diarrhea or constipation; the pallor, fretfulness and emaciation present in these cases are symptoms which call for a so-called "constitutional" remedy.

In the acute digestive disturbances there may be a predominance of local or of general symptoms as in the case of the infectious diseases. When vomiting is a predominating symptom *ippecac* is suggested as the remedy; in the case of a simple diarrhea with undigested food particles *chamomilla* will be thought of. The character of the stool, however, must be carefully studied in order to make an accurate prescription. Thus, frequent, large, yellow or greenish, liquid stools, expelled with gas and causing excoriation of the buttox call for *podophyllum*. Greenish stools containing mucus and curds with marked peevishness; distended abdomen and colic is a mild dyspeptic condition usually observed in teething infants and relieved by *chamomilla*. Intestinal disturbances with colicky pains relieved by pressure are helped by *colocynthis*. This symptom is elicited by observing that the infant is comforted and stops crying whenever the nurse lays it on its stomach, across her lap. In the case of *chamomilla* the child is promptly comforted by being carried around. When the stools contain mucus and blood indicating an infectious diarrhea *mercurius sol.* is indi-

cated. Other remedies beside *mercurius* present these symptoms and must, therefore, be differentiated. When there is vomiting and marked prostration *arsenicum album* comes to our mind. When tenesmus is pronounced *mercurius corrosivus* is better indicated than *merc. sol.* Cases with marked toxemia will suggest remedies like *belladonna*, *cuprum ars.*, *helleborus* and *rhus tox.*

In prescribing for the acute respiratory affections a knowledge of the pathology of the condition under treatment is necessary in order to select the proper remedies. This holds good in almost all diseases and whenever the selection of a remedy is based on drug pathogenesis instead of on subjective symptoms, the prescription is bound to be more accurate and the results will be better. Subjective symptoms and certain modalities are unquestionably useful in prescribing but their use is chiefly to differentiate between a group of remedies all of which have a similar pathological relationship to the case under consideration. This point is particularly well illustrated in the pneumonias. There are two well-known types of pneumonia, the catarrhal type or bronchopneumonia and croupous pneumonia. The remedies which are most frequently indicated and useful in the catarrhal type are those which affect chiefly the mucous membranes and set up catarrhal inflammations. The most important members of this group are *belladonna*, *ippecac*, *mercurius* and *tartar emetic.* In lobar pneumonia we must turn to a different group, namely one whose pathology corresponds more nearly with that of vascular engorgement and croupous exudation and *aconite*, *bryonia*, *iodine* and *phosphorus* come to our mind.

Belladonna is a most useful remedy in the early stages of bronchopneumonia indicated by the dry, paroxysmal cough; high fever with flushed face and cerebral excitement. Many capable clinicians believe that when *belladonna* is used early a large number of cases of bronchitis can be aborted and that the extension of the process into the finer bronchi and air cells can be arrested.

Ipecac is indicated when the catarrhal symptoms predominate and when the chest seems literally filled with mucous secretion. The cough is associated with gagging and vomiting of mucus. When the secretion accumulates in the finer tubes the clinical picture of a capillary bronchitis develops. Cyanosis gradually develops and the child is no longer able to discharge the secretion from the bronchi and the mucus collects in the larger tubes, producing coarse rattling rales. At this stage of the disease *tartar emetic* is indicated and it may still help us to pull the case through unless circulatory failure and pulmonary edema supervene.

Bryonia occupies the unique position of being the most generally useful remedy in all forms of acute respiratory disease. It causes inflammation of the bronchi, lungs and serous membranes and its symptomatology covers the most important clinical features of the majority of cases of bronchitis and pneumonia. There is a hard, deep, non-productive cough which is painful and which is made worse by talking, drinking, or bodily exertion. The child, therefore, lies quietly and resents being moved or disturbed. There is fever, headache, mild delirium, irritability and great thirst. The bowels are constipated. When pleurisy develops as a complication *bryonia* is still the best remedy for the case.

Scilla maritima is useful in the severe types of bronchopneumonia with hard, painful cough. The cough is more paroxysmal than that of *bryonia*, there is free secretion in the bronchi as indicated by an abundance of moist rales over the bases of the lungs and there is more prostration than in a *bryonia* case.

In croupous pneumonia we think of *aconite* in the first stage which is of sudden onset with chill or its equivalent; the child is excited and restless and chest symptoms may be slight or wanting. If there is cough with the characteristic blood-streaked sputum, *ferrum phos.* is the remedy of choice. Pleuritic involvement calls for *bryonia* and it may be alternated with

either *aconite* or *ferrum phos.* *Bryonia* is also indicated as soon as consolidation develops. *Phosphorus* is useful in the graver types of pneumonia with toxemia, pulmonary congestion and dyspnea; expectoration of pure blood. *Hyoscyamus* is indicated in the cerebral type simulating meningitis.

A remedy which has been of great help to the homeopath in the treatment of poliomyelitis and lethargic encephalitis is *gelsemium*. The symptoms recorded in the provings and toxicological reports of this drug are very characteristic and correspond closely with some of the leading clinical manifestations of the disease mentioned. I am sure that we have all had occasion to see the good effects of this remedy in the cases of poliomyelitis which have come under our notice. In spite of the claims made for the efficacy of immune horse serum in this disease I cannot see that they are sufficiently striking to make a better showing than homeopathic treatment.

The so-called constitutional remedy is one of the homeopathic pediatricist's chief assets. There is no longer any doubt in the minds of either school of medical practitioners that certain types of individuals are susceptible to certain diseases and that some react more strongly to certain drugs than others do. Vagotonia and sympathicotonia are terms which our old school colleagues recognize and understand but before these terms were introduced into medicine the observations made by homeopathic practitioners that certain individuals were hypersensitive to certain drugs and that small doses of such drugs administered to a susceptible individual would produce a striking drug effect were rejected as absurd and unscientific. Likewise the homeopath's insistence upon the importance of diathetic or constitutional abnormalities, or the dyscrasiæ, as we call them, was ignored. The dominant school, however, now recognizes many such dyscrasiæ, for example, exudative diathesia, lithemia, spasmophilia, scrofula and status lymphaticus.

In the homeopathic literature it has been the custom to refer to a certain type of child as representing a certain remedy.

Thus, we read of the *calcareo carbonica* baby; the *sulphur* patient; the *pulsatilla* female. This method of expressing a certain therapeutic idea has no doubt provoked mirth in the minds of those unfamiliar with the homeopathic method of drug study. However, the idea is a good one and the point which our materia medica teachers have attempted to make by this verbal formula is to present a mental picture of the clinical sphere of the drug. When they describe the *calcareo* child they draw a clinical picture of the type of child which needs *calcareo carb.*—the fair complexioned, fat infant with poor muscular tone, open fontanel; delayed dentition; sweating about the head; large belly; large, pale constipated or sour dyspeptic stools. There should be no question in the mind of anyone that a remedial agent which will improve the calcium metabolism of such an infant will be the best possible thing for it and calcium given in the finely-divided form of a homeopathic trituration stands a better chance of doing this than crude doses of lime salts.

The clinical type described under the *iodine* syndrome represents glandular atrophy and marasmus. The keynote symptoms of *graphites* and *sulphur* will be encountered in infants presenting the exudative diathesis. The endocrinologist has gone perhaps further than the homeopath in his classification of individuals into certain types or “tropes” to indicate which one of the organs of internal secretion is at fault. In a case presenting distinct clinical evidence of glandular insufficiency the administration of the proper gland substance should be expected to give better results than medicines. This, however, is not the case excepting in hypothyroidism and we must still look to the constitutional remedy for help in many chronic and nutritional disorders.

CHAPTER IV.

INFANT FEEDING.

Owing to the fact that by far the largest number of deaths occurring during infancy can be traced to disturbances of nutrition and diseases of the gastro-intestinal tract, infant feeding occupies the most conspicuous place in the specialty of diseases of children.

Infant feeding may be divided into *natural*, or *maternal feeding* and *artificial*, or *bottle-feeding*. Nature has intended that the infant be fed at its mother's breast and whenever maternal feeding can be carried out it should be encouraged to the fullest extent. Artificial, or bottle-feeding is not a method of choice but one of necessity. Unfortunately a large proportion of all infants cannot enjoy the advantages of maternal nursing for which reason it becomes necessary to institute artificial feeding. Under these circumstances it is our duty to decide upon a proper substitute for woman's milk and to give the mother instructions in the preparation of the food as well as in the manner of feeding the same, namely, the amount to be given at a feeding and the number of feedings necessary in the twenty-four hours. Before, however, taking up the subject of feeding we will first discuss the underlying principles above referred to.

ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE TRACT.

The *mouth* is dry during early infancy on account of the absence of salivary secretion. As the teeth begin to erupt saliva is secreted, at times excessively, causing the infant to "drool." The salivary glands are anatomically developed in early life and ptyalin can be demonstrated in the salivary secretion; however, owing to the fact that the babe receives its food in liquid form there is no need for saliva at this time of life.

The stomach is in an immature state at birth, the fundus being poorly developed. The lesser curvature occupies a more horizontal position than after the child walks. The pylorus, which is in the median line and covered by the liver in the fetus, gradually moves over toward the right as the child grows older.

Vomiting occurs readily on account of the immature development of the sphincter muscle at the cardiac end. Dilatation of the stomach readily occurs from prolonged overfeeding, and hypertrophy with *hyperperistalsis* develops early in cases of congenital pyloric obstruction.

The gastric capacity is about one ounce in the new-born and there is a gradual and progressive increase at the rate of one ounce per month so that at one month the capacity will be two ounces; at three months four ounces; at six months seven ounces. After the sixth month the increase is not so great and we may allow nine ounces for a nine months' old infant and ten ounces at one year.

The gastric juice of the infant is identical with that of the older child, with the exception that there is a smaller percentage of hydrochloric acid. Free hydrochloric acid cannot be demonstrated when the infant is on a diet of cow's milk because of the complete union of the acid with the casein. However, when barley-water is used as a test meal, the reaction for free hydrochloric acid can be obtained.

Milk is coagulated within a few minutes after entering the stomach by the rennin of the gastric juice. The whey is squeezed out from the curd and so the stomach begins to discharge the liquid portion of its contents before the child has finished his meal. A normal acid reaction of the gastric contents is necessary for the digestion to progress favorably, as the pylorus remains closed as long as the contents of the pyloric end of the stomach are alkaline. The addition of alkalies to the food in sufficient amount to make it excessively alkaline will therefore delay rather than assist gastric digestion. It will also neutralize the hydrochloric acid of the gastric juice

and deprive the child of the benefits of this useful secretion, which possesses both germicidal and denaturizing properties, destroying bacteria and their toxins in the food to a great extent, and preventing foreign proteins from setting up anaphylactic reactions.

The average emptying time of the stomach is about three hours. The time required for the digestion of a meal depends to a great extent, however, upon the character of the food, far more so than upon the amount taken. Woman's milk is more quickly digested and leaves the stomach sooner than a meal of modified milk. The carbohydrates are quickly ejected from the stomach into the duodenum. In fact this occurs as soon as the milk has coagulated, as has been stated above. As a result of this early passage of the whey into the intestine the stomach is really not distended to the full capacity of the amount taken at each meal, provided the infant has not been fed too rapidly.

After the carbohydrates, the proteins leave the stomach, usually at the end of two hours; the fats leaving last. The interval between feedings must, therefore, be gauged according to the composition of the food rather than according to the quantity given at each feeding or the age of the child. A food high in carbohydrates but poor in protein and fat leaves the stomach in a short time. High protein mixtures require a moderate length of time to digest—from two to three hours—while milk rich in fat, whether breast milk or a modified cow's milk, requires the longest periods for complete gastric digestion and should, therefore, be given at four hour intervals.

The Intestines. After the food passes through the pylorus it enters the duodenum where intestinal digestion begins. The chyme is acid when first entering the duodenum, but is neutralized and rendered alkaline by the pancreatic secretion. The pylorus remains closed so long as the duodenal secretion is acid; in this way the flow of chyme from the stomach is automatically regulated and is more or less intermittent.

The digestive function of the pancreatic secretion is three-fold, namely: proteolytic, amylotic and fat-splitting. The ferments are all present at birth but pancreatic activity increases with the growth of the infant.

The pancreas does not begin to secrete until stimulated by the action of the secretion present in the intestinal mucosa. When the acid chyme reaches the duodenum it causes secretin to be set free. This hormone is carried to the pancreas by the blood current and activates the pancreatic cells.

The biliary secretion contains a ferment which changes a pro-enzyme of the pancreatic juice into steapsin. The importance of bile in the digestion of fat is thus explained. The yellow color of the normal breast-fed stool is due to the presence of bilirubin. In dyspeptic conditions bilirubin is oxidized into biliverdin giving the stool a green color.

The succus entericus secreted by the small intestines contains specific ferments for converting disaccharids such as sugar of milk, maltose, and cane sugar into monosaccharids.

THE STOOLS.

The newborn infant expels a dark, greenish, tarry substance of semi-solid consistency from the bowels which is called *meconium*. Its composition is biliary and intestinal secretion from which most of the moisture has been absorbed and which has accumulated in the gut during fetal life. Epithelium, hairs and vernix caseosa are also present showing that the fetus swallows amniotic fluid. On the third day the discharges from the bowels become thinner, contain more mucus and assume a brownish-green color. This is identical in appearance and composition with the *starvation-stool* observed when a child is fed for several days on a diet containing no solid constituents such as barley-water, broth or tea.

FACTORS INFLUENCING THE CHARACTER OF THE STOOLS.

The diet is largely responsible for the appearance and character of the stools. The state of the digestion also affects their appearance and we must therefore differentiate between physiological and pathological deviations from the average normal. A young infant may have from three to four stools daily, while an older one will normally have from one to two in 24 hours.

The frequency of the stool is greater in breast-fed than in bottle-fed infants and the consistency may be normally much thinner. A thin stool containing some mucus may simply indicate that the milk is poor in fat. The consistency of the stool is therefore largely dependent upon the amount of fat in the food. When the infant is fed upon a food containing fairly large amounts of protein and carbohydrate but a low percentage of fat the stools are usually soft and salve-like. When the food is rich in fat and low in protein the stools may be soft or semi-liquid and contain curds. In cases of overfeeding with both casein and cream the stools become large and formed, of a putty-like consistency and grayish color, due to the formation of calcium soap in the intestinal tract. The soaps produced by the union of fatty acids with the calcium salts of the intestinal secretion, are dry and insoluble and therefore produce troublesome constipation.

The color of the breast-fed stool is an orange-yellow due to the presence of unchanged bilirubin. In infants fed on cow's milk the stools are of a paler shade. Sometimes they become almost colorless, as a result of the reduction of bilirubin into hydrobilirubin or urobilinogen, through bacterial action. This is most frequently seen in fat indigestion with constipated stools. When high protein and low fat formulæ are fed, the stools may have a brownish-yellow color. Barley and maltose preparations give the stool a brownish color. In dyspeptic conditions resulting from fermentation of the sugar of milk in the food, the stool is usually green.

The reaction of the stool is determined by means of strips of red and blue litmus paper moistened with water. It is acid in the breast-fed infant as a result of the fermentation changes taking place in the intestinal canal. This is favored by the high sugar (lactose) and low protein content of the mother's milk. In bottle-fed infants the stools are alkaline as a result of putrefactive changes in the intestine. The reaction may be made acid by feeding high fat and low protein formulæ and by means of high sugar formulæ. There is, however, always danger of the fermentation process exceeding the normal limits in artificial feeding.

In the lower portion of the small intestine and in the cecum of the breast-fed infant the *bacillus lactis aerogenes* and the *bacillus bifidus* predominate. The latter is most prevalent in the colon. Both are saccharolytic, converting lactose into lactic acid. The *bacillus bifidus* is Gram positive in its behavior to stains.

In the artificially fed infant the colon group of bacteria predominates. They are proteolytic, although in a medium consisting chiefly of carbohydrate they may set up fermentative changes. The colon bacillus is Gram negative.

Aside from the higher protein percentage of cow's milk the putrefactive changes which take place in the albuminous intestinal secretion are also responsible for the bacteriological difference between the intestinal tract of breast-fed and artificially fed infants. The protein-rich artificial food stimulates intestinal secretion to a greater degree than does woman's milk. The alkalinity of the secretion favors putrefaction as does also the relatively higher calcium content of cow's milk. Several factors, therefore, are operative in bringing about these proteolytic changes.

ABNORMAL CONSTITUENTS IN THE STOOLS.

Curds are one of the most important abnormal constituents of the stool and are usually associated with an excess of moisture and mucus, so that the stool becomes too soft or liquid. Such

a loose stool is typical of indigestion although in breast-fed infants several stools of this character may be passed daily without any evidence of dyspepsia.

The majority of curds indicate fat indigestion. They are composed of neutral fats and calcium soap, resulting from the combination of the fatty acids of the food with the mineral bases present in the intestinal secretion. Fat curds may be soft and oily, imparting an oily stain to a piece of unglazed paper when crushed upon the same, if they contain an appreciable amount of neutral fat. When they are composed mainly of calcium soap they are dryer and more brittle. They always contain some adventitious protein matter which may give them a tough consistency. The large, dry, hard fecal masses encountered in constipation from overfeeding are a good example of calcium soap stools.

Casein, or protein curds, are far less common than fat curds. They only occur when unboiled cow's milk is used and so can readily be overcome by boiling the milk. Casein curds are tougher than fat curds and are hardened by the action of formaldehyde. The curd should be placed on a piece of filter paper and some formaldehyde poured over it. If it is essentially a protein curd its consistency will promptly be changed by the reagent.

Chemical Examination of Curds. A protein curd will respond to the usual tests for protein such as the Xanthoproteic reaction and Piotrowski's reaction. It should be remembered, however, that a considerable proportion of the make up of curds and elements in the stool must be attributed to the albuminous intestinal secretion and to bacteria, and for this reason all curds, whether primarily resulting from undigested or unassimilated fat or casein, will be contaminated with extraneous protein.

A washed portion of the curd is placed in a test tube, dilute nitric acid is added and the contents of the tube boiled. All proteins are dissolved by the action of such a hot acid solution, and the solution assumes a yellow color. When it has cooled,

a strong alkali, such as sodium hydroxide solution, is added and an orange-yellow color reaction takes place. This is the *Xanthoproteic reaction*. *Piotrowski's* reaction is obtained by adding to the above solution a drop of copper sulphate and then an excess of sodium hydroxide. The reaction is a violet color, becoming darker on boiling.

Fat curds are not influenced by formaldehyde but they are melted by heat if acetic acid be added to break down the soaps and liberate the fatty acids. The latter will crystalize out on cooling. The various fat elements also exhibit certain peculiarities of staining which make their identification possible. The test is best carried out under the low power of the microscope. A small fragment of stool is placed on a glass slide, mixed with a drop of water and then a drop of dilute carbol-fuchsin stain is added and a coverglass applied. The soap particles present take on a pale rose color while the fatty acids take a deep red stain.

Neutral fats do not take the fuchsin stain but can be demonstrated by treating a specimen prepared as above with alcoholic Sudan III. With this stain the neutral fats show up as orange colored droplets. The fatty acids take on a deep red.

If a drop of glacial acetic acid be allowed to run under the edge of the coverglass and the specimen heated, the fatty acids are liberated and on cooling will appear as needle-like crystals.

This test is of value to establish the identity of a given curd and it also gives an approximate idea of the amount of fat in the stool. In normal, well digested stools only a trace of soap and fatty acids will be found. The younger the infant, however, the less complete the assimilation of fat even under normal conditions. The presence of neutral fats indicates duodenal indigestion from excessive peristalsis or sugar dyspepsia, or it may be an early sign of fat overfeeding. Excess of soaps in the stool indicates chronic fat indigestion, usually as a result of overfeeding. Normally about 90 per cent of the fat is assimilated.

Bile is present normally in the breast-fed stool in the form of bilirubin, which gives the stool its bright yellow color. Under artificial feeding the biliary constituents are often changed. In constipated stools bacterial reduction takes place and most of the bilirubin has been changed to urobilinogen, so that the stools are much lighter in color. In fermentation disturbances the bilirubin has been oxidized to biliverdin, giving the stool a green color. When exposed to the air a loose stool which was yellow when passed, oxidizes to a green color.

Bacteriological Examination. It has been stated that the breast-fed stool is Gram positive while the stool of the bottle baby is chiefly Gram negative. In the latter the colon bacillus predominates and this organism may act either as a fermentative or as a putrefactive agent.

Among the pathogenic organisms demonstrable in the intestinal tract the most important are the dysentery bacilli of which there are several types. These organisms may at times be demonstrated in the stools of healthy infants but when they are found in a case of ileocolitis and give the agglutination reaction with the blood of the patient, they may properly be considered as an infective agent. Unfortunately the cultivation and identification of the dysentery group requires special laboratory facilities and technical skill and therefore cannot be carried out in general practice. Sometimes they can be identified as Gram negative intracellular bacilli (Grulee).

The gas bacillus of Welch is looked upon by Kendall as the etiological factor in certain cases of infantile diarrhea and the interesting point in connection with this type of infection is that he considers the lactic acid bacillus, given either in the form of buttermilk or in culture, a therapeutic specific. Holt considers its presence in diarrheal cases purely accidental. Its identification is simple and therefore clinically practical. A small portion of the suspected stool is thoroughly mixed with several cubic centimeters of milk in a test tube, the tube is placed in a water-bath of cold water, the water slowly brought

to the boiling point and allowed to boil for three minutes. All bacteria are killed by this heat excepting the spores of the gas bacillus. After incubating the tube at body temperature for twenty-four hours the gas bacillus, if present, liquifies the major portion of the casein and the residue assumes a pinkish color and contains small gas bubbles. The odor of butyric acid is also evolved. The bacillus is a thick, short rod with rounded ends and is Gram positive.

MATERNAL NURSING.

The physician should insist upon the mother nursing her infant whenever this is possible. A frequent error, the gravity of which does not seem to be fully realized, is to wean the babe because the milk appears to disagree with the infant. There may be spitting up, colic or loose, curdled stools as a cause for this decision. The proper regulation of the intervals of feeding and the administration of a little hot water before each feeding, will overcome these apparent faults of the food. Again, the child may be weaned because the breast-milk is insufficient in quantity, not worth while, or too poor in quality to properly nourish. Here a resort to mixed feeding, administering an ounce or two of some appropriate modified milk formula after each feeding will give the infant practically all of the advantages of maternal nursing.

There are, of course, definite contraindications to maternal nursing. The mother with open tuberculosis cannot nurse her babe on account of the drain upon her own vitality and the danger to the child. In poorly compensated heart affections the mother should always be relieved from the task of nursing her own babe. In Bright's disease it will depend largely upon her ability to stand the strain. In eclampsia nursing is contraindicated.

The syphilitic mother should by all means nurse her baby if possible. Under no circumstances should the babe of a syphilitic woman be given to another woman to nurse.

In acute affections of short duration nursing need not be interdicted but in sepsis or typhoid fever the infant should be weaned.

The infant should be put to the breast as soon as the mother has recovered from the effects of the labor. This may be done twice daily and in the interim, at four hour intervals, a few teaspoonfuls of tepid water may be given.

The secretion found in the breast before lactation is known as *colostrum*. This is a highly albuminous substance with laxative properties and appears to be essential to the welfare of the newborn. Infants that are denied the breast from the very beginning usually do poorly and are difficult feeding cases.

By the third day the flow of milk is established and the infant should be nursed every three hours during the day and every four hours after 6 P. M., making seven feedings in the twenty-four hours.

After the infant is two months old the midnight feeding may be omitted, making six feedings in the twenty-four hours. After the fifth month the intervals between feedings should be lengthened to four hours, so that there will be but five daily nursings.

The duration of nursing is normally about fifteen minutes. It has been shown that the infant gets one half the contents of the breast during the first five minutes. During the second five minutes it gets an additional quarter of the total amount and during the third five minutes, the balance. If the child is not a vigorous nurser it may be allowed twenty minutes, but not longer. A child may fall asleep during nursing, from feebleness; under these conditions it may be nursed more frequently for a while until it has gained strength. If the quantity is insufficient it will fret and cry when taken from the breast.

If the child's nose is stopped up it lets go the nipple every few seconds in order to get its breath. This may also occur from faulty position whereby the breast interferes with the

child's breathing. A sore mouth will cause the child to let go the nipple and cry with pain.

The nipple should be washed before and after each nursing with a saturated solution of boric acid, but it is not advisable to wash out the baby's mouth between feedings. The breasts should be given in rotation. When the milk becomes scanty it may be advisable to give both breasts at a feeding but this should only be done if the infant drains both completely. Under these circumstances a bottle will usually be necessary between feedings.

Indications for Mixed Feeding.—When the infant fails to gain, or actually loses weight and shows signs of hunger it is not getting a sufficient amount of food. Most frequently the quantity is deficient; at times, however, the milk of poor quality may account for this undernutrition. In both instances mixed feeding will have to be resorted to.

If the examination of the milk shows it to be *deficient in solids*, particularly in fat, half an ounce of modified cow's milk may be given after each feeding. Under these circumstances it will be best to use a 10 per cent top-milk diluted with two parts of a 5 per cent solution of sugar of milk.

Deficient quantity is best determined by weighing the child before and after each feeding. This simple procedure gives an approximate idea of the average amount of milk which the infant obtains at a feeding. The deficiency can then be made up by giving the child the proper amount of modified milk after each nursing. Thus, if a two months old infant only gets two ounces at each feeding we should give it one to two ounces of modified milk according to its weight, after each nursing. The best formula to use under these circumstances is the regular one-third whole milk diluted with two parts water and 5 per cent sugar added to the total quantity.

The reason for giving the bottle after the feeding is to insure thorough emptying of the breast. Unless the child is hungry it will not nurse vigorously and so the breast will lose the physiological stimulus necessary to keep up the process of lactation.

Alternating the breast with the bottle is more likely to result in the child weaning itself. The average infant will grow to prefer the bottle to the breast because it gets the food with so much less effort. Only when the breasts begin to fail in their function should both breasts be given at one feeding and a bottle at the next.

INDICATIONS FOR WEANING.

If the child is too weak to nurse, the milk should be pumped from the breast and fed with a spoon or medicine dropper. Weaning should always be put off as a last resort. Even if the infant only gets the benefit of the colostrum and two or three weeks of partial breast feeding it will have had help at a time when artificial feeding is most difficult to institute successfully.

The imperative indications for weaning have already been mentioned. Menstruation and pregnancy are not necessarily indications for immediate weaning, so long as mother and babe are doing perfectly well.

Cracked nipples may cause the mother great suffering, but a nipple shield will overcome this trouble. Abnormally formed nipples may also be made to serve the babe with the aid of a nipple shield.

The gradual deterioration of the milk and the increasing demands of the organism for more protein and salts during the last quarter of the first year, make weaning at this time a logical matter. Should this time come in mid-summer it may be permissible to carry the child along on the breast until it is a year old, providing, of course, the mother is able to continue with the nursing. It is advisable, however, to give a feeding once daily of a strained vegetable soup, in order to supply additional mineral salts, carbohydrate and vegetable protein.

The infant should be taught to drink water from earliest infancy. This will make weaning much easier when the time arrives. Also, during the hot weather, the child may be given a few ounces of water daily with advantage.

False Indications for Weaning are dyspeptic symptoms such as vomiting, curds in the stools and colic. Usually these symptoms are promptly corrected by instituting four hour intervals when the infant has been nursed too frequently. Should the milk show an abnormally high percentage of fat we may give the babe a teaspoonful or two of warm water before putting it to the breast; also cutting down the mother's diet. In case of persistent disturbance an ounce of barley-water between feedings together with omitting sugar and fat from the mother's diet will correct the trouble.

Poor quality of the milk and deficient quantity should not lead to weaning but call for mixed feeding as described above.

The Wet-Nurse.—Premature and congenitally feeble infants are most difficult to raise on artificial food and if the mother is unable to nurse, a wet nurse should be procured. Young infants who have been badly started on the bottle and who have developed a marked intolerance for cow's milk are in imminent danger unless they can be put to the breast. When the choice of a wet-nurse is therefore imperative it is indeed a time of trouble and tribulation for both the physician and the patient unless a woman well known to either of them is available. When it is impossible to obtain a nurse who can stay in the patient's home and nurse the infant for every feeding, we may be able to get good results from having the wet-nurse come to the house three times daily. Sometimes a woman who is nursing her own babe can be gotten for this purpose. The only requirements necessary for a wet-nurse are that she be in good health and have a sufficient supply of average good milk. All other matters such as the period of lactation, whether or not she is nursing her own infant, her age, etc., are negligible.

In most large cities wet-nurses may be obtained from the various Maternity Hospitals or through Child Welfare Bureaus and Directories for Wet-Nurses. Before accepting such a professional wet-nurse a certificate of freedom from tuberculosis and gonorrhea and a negative Wasserman should be demanded.

METABOLISM IN INFANCY.

The newborn infant requires forty-five calories of food for every pound of body-weight, while the adult requires but twenty. The food requirement thus expressed in calories per pound of body-weight is called the "energy-quotient," and it furnishes a valuable guide for estimating the amount of food needed by the infant. The age of the infant and its weight, therefore, become the determining factors to decide the quantity and strength of the food to be used at the different periods of infancy. The so-called "Caloric System" of feeding is based upon these facts. There is, however, no such thing as a caloric system of feeding; the energy-quotient simply guides us in the dosage of the food and is a most valuable check against both underfeeding and overfeeding.

Metabolism in infancy differs radically from metabolism in the adult and it is necessary to have a thorough understanding of this subject before one can grasp the fundamental principles of infant feeding.

It has been estimated that the infant requires about one-sixth of its body-weight in food (woman's milk) during the first three months of its life; one-seventh during the second quarter; one-eighth during the third quarter; and one-ninth during the last quarter of the first year. Expressed in calories this corresponds to about 45 C. per pound of body-weight for the first quarter, 44 C. for the second quarter, 42 C. for the third quarter and 40 at one year.

The explanation for this very high demand for food at birth and the gradual decline in the demand, dropping to 40 C. for each pound of body-weight at one year, 27 C. at the age of ten years, and 20 C. at the time of attainment of full growth, or adult life, is self evident when we pause to consider that the infant is a rapidly growing organism and requires food not only for the production of heat and energy and to replace broken down tissues but also for the formation of new tissue and for

the multiplication of the body cells. As the infant grows most rapidly during the first months of life its food requirements are greatest at that time. Thus during the first quarter it gains on an average of half a pound a week, thereby doubling its birth weight at the end of the fifth month. After that time it gains about a third of a pound per week and so trebles its birth weight at the end of the first year. During the entire second year it gains only as much as it did during the first five months, and thereby quadruples its birth weight at the end of the second year. The growth of the child in length takes place correspondingly with the increase in weight, very rapidly in early infancy and gradually falling off. The growth of the child, therefore, is most active during early infancy. When the full adult type of the sex is attained, the height and weight of the individual become stationary. The amount of food required at the different ages, therefore, is largely dependent upon the rate at which the individual is growing.

Another factor accounting for the high food requirements of the infant in comparison with an older child is the relatively greater body surface which the infant presents, for which reason the loss of body heat is proportionately higher in a small individual than in a larger one. The great activity of the infant, especially cardiac and respiratory, and the larger amount of glandular energy expended in the digestion of the large amount of food needed daily are other factors influencing the food requirements.

An important difference between the child-type of metabolism and the adult-type is that the adult has attained "nitrogen-equilibrium," while the child retains a large portion of the nitrogen taken in its food. In the adult, under normal conditions, as much nitrogen can be recovered from the urine and feces as was ingested with the food. The child, however, retains a large part of the nitrogen, utilizing the same in building up the body tissues.

Food values are measured in calories and the great calorie

(C) is employed in our calculations in dietetics. It represents the quantity of food in grams which will give off a sufficient amount of heat when burned in the calorimeter to raise one kilogram of water one degree centigrade. Since the process of oxidation enters so largely into the phenomena of metabolism this method of estimating the food, or fuel value of the various foodstuffs is both logical and practical.

The fuel value or caloric value of the different foodstuffs is as follows:—

One gram of protein	4.1 C.
One gram of carbohydrate	4.1 C.
One gram of fat	9.3 C.

From the above figures it is possible to compute the caloric value of the various foods entering into the child's dietary and if we know how many calories the child needs daily we are in a position to check up its diet in a scientific manner.

The following table gives the caloric value per ounce of a number of the more important foods employed in infant feeding:—

Woman's Milk	20 C.
Cow's Milk, 4 per cent fat.....	20 C.
Cream, 16 per cent fat.....	54 C.
Top-milk, 10 per cent fat.....	33 C.
Top-milk, 7 per cent fat.....	30 C.
Skimmed Milk	10 C.
Carbohydrate (the sugars)	120 C.

The following table will be found useful in computing the number of calories present in the diets for older children. Some of these figures can be easily memorized if we remember, for example, the fact that one egg, a cubic inch of butter, a slice of bread weighing one ounce and a medium sized potato, have about the same caloric value:—

One egg	75 C.
A piece of butter a cubic inch square.....	75 C.
A slice of bread, weight one ounce.....	75 C.
A medium sized potato.....	75 C.
Olive Oil, one teaspoonful.....	75 C.

Sugar or other carbohydrate (rice, etc., dry)—120 C. per ounce. Lean meat equals about 30 C. per ounce.

Protein Metabolism.—The protein molecule contains all of the elements needed for cell growth and cell multiplication. Growth is therefore dependent upon a sufficient amount of protein in the food. Carbon, hydrogen and oxygen are found in all foods, namely in carbohydrates, fats and proteins. The protoplasm of the cell, however, requires beside these three elements, nitrogen and phosphorus and these are found in the casein of the milk. Casein also contains a large amount of calcium which adapts it especially to the food requirements of the infant.

During digestion the protein molecule is broken down into the elementary amino-acid groups of which it is chiefly composed, by the action of the digestive ferments and these cleavage products are absorbed into the general circulation and carried to the tissues where they are again built up or synthesized into the specific proteins of the various organs.

The infant retains most of the nitrogen taken in the food because it utilizes the protein cleavage products in building up its tissues. Some nitrogen appears in the urine indicating that some of the radicals from the protein molecule have been used for heat production, or dynamogenic purposes. Nitrogen may also appear in the urine in the form of ammonia products, resulting from an acidosis. The addition of carbohydrate to the food increases nitrogen retention if it is not pushed to the point of setting up a diarrhea.

The feeding of milk mixtures containing a high percentage of protein (casein) does not produce metabolic disturbances such as fever or uremic manifestations nor does it upset the digestion so

long as there is not too much fat or sugar in the mixture to cause a primary digestive disturbance. It has been previously stated that the majority of curds found in the stools of bottle-fed infants consist of fat products and not of undigested casein; also that when true casein curds do occur they can be controlled by boiling the milk. A formula containing a high casein percentage, such as Finkelstein's "Eiweissmilch" is actually beneficial in fermentative dyspeptic conditions. Proteins stimulate the intestinal secretion and this secretion being alkaline, neutralizes the acids resulting from the fermentation. High protein in the food also inhibits fermentation. Under ordinary conditions, however, it is not advisable to administer larger amounts of casein than are found in the milk dilutions generally employed in infant feeding.

Fat Metabolism.—The digestion and the assimilation of fat is not as complete as that of protein. The newborn passes a large amount of unassimilated fat in its stools in the early weeks of life but as it grows older fat digestion becomes more complete and there is an absorption of about 90 per cent of the fat present in the food. The normal bottle-fed infant also assimilates about 90 per cent of the ingested fat.

The consistency of the stool depends largely upon the amount of fat residue which it contains. According to the investigations of Holt (*American Journal of Diseases of Children*, April, 1919 and June, 1919) the fat of the stools of normal breast-fed infants averaged 34.5 per cent of the dried weight. In normal bottle-fed infants the percentage averaged 36.2. The soap per cent of total fat averaged about 73, being highest in the constipated stools. The soap is produced by the combination of unassimilated fatty acids with the alkaline mineral salts of the intestinal secretion. The soap of the constipated stool is dry and insoluble owing to the predominance of calcium salts.

The formation of calcium soap stools is favored by milk overfeeding, as a result of which the child receives more protein and fat in twenty-four hours than it can digest or assimilate.

The excess of protein stimulates intestinal secretion and favors putrefaction changes in the gut, and this in turn favors soap-stool formation. Czerny has designated the clinical picture resulting from prolonged overfeeding, "Milchnahrschaden." There is loss of weight, fretfulness, pallor, tympanites and hard, dry, grayish, offensive stools together with excess of ammonia in the urine. A diet low in protein and high in carbohydrate, such as woman's milk, tends to diminish soap formation. Foods like Finkelstein's "Eiweissmilch" favor the formation of soap stools, owing to the presence of a large amount of protein and calcium salt entering into its composition. Peristalsis is thereby checked and diarrhea overcome.

The long continued feeding of excessive amounts of fat may lead to the development of an acidosis. This is indicated by the presence of an excess of ammonia in the urine and of acetone and diacetic acid. A preceeding malnutrition, resulting from the overfeeding, favors the development of acidosis. The loss of alkaline salts through the increased intestinal secretion, which combine with the fatty acids in the stool and are thus excreted, leads to an increased ammonia production in the tissues in order to neutralize the urinary acids. Therefore, if through a disturbance of fat metabolism, oxybutyric acid and diacetic acid are formed in the tissues acidosis promptly develops because the organism has lost its power of neutralizing these acids in the blood plasma. The tolerance to fat, of course, varies widely in different individuals but the dangers of fat overfeeding must always be borne in mind.

Carbohydrate Metabolism.—The carbohydrate found in milk is lactose, or milk sugar. Lactose is a disaccharid, as are the other sugars used in infant feeding. After the lactose reaches the intestines it is acted upon by an enzyme, invertin, which splits it into the monosaccharids dextrose and galactose. Cane sugar is acted upon similarly and is split into dextrose and levulose. Maltose is the end product of starch digestion; one molecule of maltose is split into two molecules of dextrose

through the action of invertin. Maltose is also split in the stomach to a slight extent by the hydrolytic action of the hydrochloric acid of the gastric juice.

Starch is a polysaccharid; it is hydrolyzed both by the ptyalin of the saliva and by the amylopsin of the pancreatic juice. Starch digestion takes place mainly in the intestine. The starch is first converted into a soluble form, or amyloextrin. This is further split into dextrin and malt-sugar, or maltose. The final product is maltose.

A large percentage of the sugars ingested with the food is lost through fermentation. Lactose is most readily affected by the intestinal bacteria, undergoing lactic acid fermentation which gives the stool of the breast-fed infant its characteristic sour odor. In bottle-fed infants this fermentation is likely to exceed the limits which occur in natural feeding and an acid diarrhea frequently results from the fermentation of the lactose. If the fermentation is not promptly controlled serious disturbances may result. Maltose does not ferment as readily as lactose while cane-sugar does not undergo lactic acid fermentation at all. Dextrin and starch exert an inhibiting action over intestinal fermentation and are therefore used to "correct" this tendency in the bottle-fed infant. High percentages of casein in the food also tend to inhibit fermentation. The whey of the milk, on the other hand, perhaps due to the presence of sodium and potassium salts, intensifies the process.

The infant presents a higher sugar tolerance than the adult and its metabolic needs for carbohydrates are relatively higher. Carbohydrates furnish body heat by undergoing combustion with the production of carbon dioxide and water. They also increase protein metabolism (retention) and are essential to the combustion of the fats. When the blood-sugar falls below a certain level, products of incomplete fat oxidation appear in the blood. These are oxybutyric acid, diacetic acid and acetone. A state of acidosis may supervene. Carbohydrate is therefore an essential food-stuff and cannot be entirely replaced by protein or fat.

Gain in weight is more influenced by carbohydrates than by any other food-stuff because carbohydrates exert the dual role of being eventually converted into fat in the organism if taken in sufficient amount and of favoring the retention of water in the system. The carbohydrate which is not oxidized is deposited in the liver and muscles in the form of glycogen; a certain proportion is converted into fat. The chief nutritional function of the carbohydrate, however, is to supply heat and energy, to spare the body proteins and favor the complete combustion of the fats.

The Mineral Salts in Metabolism.—The mineral salts cannot be looked upon in the light of food-stuffs but nevertheless their presence in the food in normal amounts is essential to the welfare of the organism. Inorganic salts are necessary to maintain the normal alkalinity of the blood plasma which is in constant danger of being robbed of its alkalinity by the acids formed in the body during the metabolism of proteins and fats. The growing organism also requires large amounts of calcium for the development of the osseous system while iron is needed for the blood. As a rule, a sufficient amount of mineral salts is found in the food ingested; disturbances in the metabolism of these salts result from digestive derangements or from a nutritional disorder rather than from a deficient supply.

Woman's milk contains about 0.2 per cent of mineral salts while cow's milk contains about 0.7 per cent. From this it will be seen that even when cow's milk is diluted as it usually is in infant feeding, the infant receives a little more mineral matter in the artificial food than in the woman's milk.

Sodium chloride promotes the retention of water in the system; it is perhaps the most important constituent of the blood plasma. The action of potassium is essentially that of sodium. Potassium is found freely in all vegetables and fruits and the alkalinity of the tissues is largely maintained by the salts ingested with the food.

Calcium is normally retained in the system to supply the

tissues which require it in their growth and metabolism. Under some conditions, however, more calcium is excreted from the body than is retained and we then speak of a negative calcium balance. The chief seat of calcium excretion is the intestine; when an acid reaction of the intestinal contents exists the normal resorption of calcium is interfered with. This is especially the case when the infant is fed with high-fat formulæ, the fatty acids of the stools combining with the alkaline calcium salts and forming an insoluble calcium soap which is excreted by the bowels. Calcium deficiency is especially noted in rickets and in tetary. Cod liver oil has been shown to increase calcium retention in rickets but the question arises whether this effect is due to the fat itself or to the presence of a fat-soluble vitamin which influences the rachitic process favorably. The unfavorable action of an acid intestinal tract is not limited to a disturbance of calcium balance alone. In order to meet the abnormal condition resulting from fat overfeeding the intestinal secretion is augmented and in this manner the system is drained of its alkalis. A compensatory increase in ammonia production takes place and there is an increased output of urinary ammonia. Acidosis may result if the condition is not corrected in time.

ARTIFICIAL FEEDING.

In looking about for a substitute for breast feeding we naturally turn to cow's milk as this is the only fresh milk which is commercially available. Proprietary infant foods containing milk are made from cow's milk; those which do not contain milk are not complete foods but milk modifiers intended to be added to cow's milk either for the purpose of rendering the same more digestible, or to add to the food value (carbohydrate) of the milk, or for both of these purposes.

Cow's milk cannot be successfully used as an infant food in its natural state; it must therefore be modified in order to overcome certain of its physical and chemical properties. Cow's

milk whose composition has been so altered as to conform to the digestive function of the infant and to its nutritional needs is spoken of as "modified milk." This modification is usually accomplished by diluting the milk with water in order to reduce the amount of casein present in cow's milk and by the addition of sugar (sugar of milk, cane sugar or a proprietary malt sugar preparation) to raise the percentage of carbohydrate in the mixture. Cream is also added at times and alkalies (lime water, bicarbonate of soda) have been used to render the food alkaline.

Cow's milk differs both physically and chemically from woman's milk in many important particulars. There is a biological reason for this difference (Chapin). The cow is herbivorous and its food is digested mainly in the stomach. The calf is therefore supplied with a milk which coagulates into a tough curd in the stomach and which requires the same conditions for its digestion as for the digestion of the food of the adult animal. Man in omnivorous and gastric digestion is but a preparatory step to intestinal digestion; consequently the infant's stomach must receive a food corresponding to a thoroughly masticated meal and cow's milk, in order to be acceptable, must be modified accordingly. Furthermore, as Bunge has shown, the chemical composition of the milk of the species corresponds in its protein and mineral salt content with the rapidity of growth of the young of the species. The more rapid the growth, the higher the content of these elements. The infant grows proportionately much slower than most other mammals; consequently woman's milk is poorer in protein and mineral salts than cow's milk.

The following table shows the differences in the percentage composition of woman's milk and cow's milk:

	<i>Woman's Milk</i>	<i>Cow's Milk</i>
Fat	3.5 per cent.....	4 per cent
Protein	1.5 per cent.....	3.5 per cent
Sugar	7.0 per cent.....	4.5 per cent
Salts	0.2 per cent.....	0.75 per cent

A perusal of this table shows at a glance that there is a decided difference in the percentages of the components of these two types of milk. The fats are about equal but the protein is more than twice as high in cow's milk than in woman's milk while the percentage of sugar is much lower. It is therefore a reasonable procedure to modify the milk of the cow by diluting it with water in order to reduce the amount of protein and then add sugar to bring this component up to the amount found in woman's milk. Some cream must also be added for the purpose of restoring the amount of fat which was cut down by the dilution of the milk. By this method of modification cow's milk can be so changed as to make it correspond approximately with woman's milk. Clinical experience, however, has taught us that milk thus modified does not always agree with the infant's digestion or give the best results from the standpoint of gain in weight. As a rule, a young infant requires a weaker formula while an older one requires a stronger one. Attempts to regulate the percentage of fat, protein and sugar in the food according to indications furnished by the infant's digestion, stools and gain in weight led to the development of the so-called "percentage method" of infant feeding. The percentages of fat, protein and sugar found in woman's milk were used as a standard and these percentages were varied according to the needs of the case.

Owing to the fact that this method was primarily based upon a misconception concerning the digestibility of the casein of the cow's milk it has not worked out as well clinically as was expected. Experience has taught us that the fat of cow's milk causes digestive disturbances more frequently than the casein and that artificially fed infants cannot take as high a percentage of fat in their food as is found in woman's milk. Dilutions of whole milk give better results ordinarily than milk and cream mixtures or top-milk dilutions.

The most important question to determine in artificial feeding is the amount of food which the infant requires in twenty-four

hours. Any method of feeding to give good results must be based on caloric requirements. With this thought in mind the so-called "caloric method" has been evolved.

By the caloric method the amount of food is calculated on the basis of the infant's caloric requirements according to its age and weight. All that is necessary is to determine the number of calories required by the infant in twenty-four hours and then compute the amount of milk and sugar necessary to furnish these calories. One of the chief advantages of using whole milk dilutions as the basis of the milk formula is the simplicity of this method of feeding. With proper dilution the modification of the milk to suit the infant's digestion is usually accomplished with little difficulty. Furthermore, the mother or nurse will not be burdened with instructions which are difficult for them to understand and with directions requiring much time and labor so that their many other duties are too much interfered with. The importance of avoiding complicated methods in the home modification of the infant's food cannot be exaggerated. It is true, special formulæ such as top-milk dilutions and milk and cream mixtures are frequently required in special instances. However we are likely to see our best efforts defeated if we give the mother duties to perform which she may not be able to carry out. There are milk laboratories in most large cities where special formulæ can be prepared for those who can afford this luxury and where special foods can be obtained if sickness requires their employment. The chief dangers, however, of attempting to carry out complicated methods in the home are that the food may be improperly prepared and that the mother may become discouraged and turn to the easiest remedy at hand, namely, condensed milk or some one of the proprietary foods.

Caloric Requirements in Infancy.—At birth the infant requires 45 calories per pound of body weight. As it grows older these requirements gradually fall so that at the end of a year it needs about 39 calories per pound of body weight. At

two years the requirements are about 36 calories; at ten years 27 and for an adult 20 calories per pound of body weight. While the caloric requirements in the normally growing infant therefore gradually decrease, the underweight and the premature infant on the other hand, require a relatively greater amount of food than the normal infant. Such infants may need from 50 to 60 calories per pound of body weight in order to make them gain. Unfortunately the digestion is so weak in these cases that unless they can get these calories in the form of woman's milk they will be unable to digest the food. We are not simply called upon to furnish the infant with the proper number of calories but we must give them in the form of a well balanced food in which the fat, protein, carbohydrate and mineral salts are in a digestible and available form. Furthermore, the food must also contain the necessary vitamins without which normal nutrition and metabolism are impossible. If they are not present in the milk in sufficient amount they must be supplied from other sources.

The following table gives in a convenient form the number of calories required at the different months and the average weight of the infant at these months:

CALORIC REQUIREMENTS AND WEIGHTS ACCORDING TO AGE.

At birth	7.5 lbs.	45	Calories	per lb.	Total,	337 C.
1 month	9.0 "	45	"	"	"	400 C.
2 mos.	11.0 "	44	"	"	"	480 C.
3 "	12.5 "	44	"	"	"	550 C.
4 "	14 "	42	"	"	"	590 C.
5 "	15 "	42	"	"	"	630 C.
6 "	16 "	42	"	"	"	670 C.
7 "	17 "	40	"	"	"	680 C.
8 "	18 "	40	"	"	"	720 C.
9 "	19 "	40	"	"	"	760 C.
10 "	20 "	39	"	"	"	780 C.
11 "	21 "	39	"	"	"	820 C.
12 "	22 "	39	"	"	"	860 C.
2 yrs.	28 "	36	"	"	"	1000 C.

The Digestibility of Cow's Milk.—Were it possible for the infant to digest and assimilate cow's milk as readily as woman's milk artificial feeding would present very few difficulties. Cow's milk, however, differs from woman's milk not only in the proportion of fat, protein and carbohydrate but there is also a difference in the digestibility of the fat and protein of these milks. Clinical experience has taught us that the young infant will not, as a rule, tolerate cow's milk unless it has been well diluted. After the infant has taken the diluted milk for a time, tolerance is established and the food can then be gradually made stronger. We therefore, as a rule, begin feeding milk which has been diluted with two parts water. After the second month equal parts of milk and water are usually well tolerated; from the fifth to the ninth month, two parts milk to one part water may be given.

The Proteins of Cow's Milk.—The principle protein of milk is *casein*. Casein is a nitrogenous compound which is insoluble in water and represents about 3 per cent of the milk protein. Lact-albumin is another protein found in cow's milk; it is soluble in water and furnishes one-half per cent of the proteins. The casein of cow's milk coagulates into a firm curd after reaching the stomach through the action of the milk curdling ferment in the gastric juice. Mother's milk produces a fine, flocculent curd which is readily digestible. The casein cannot alone be blamed, however, for the digestive disturbances resulting from artificial feeding. The fat and sugar must also be taken into consideration. Should casein indigestion occur as shown by the vomiting of tough curds or colic with the presence of the characteristic tough, bean-like curds in the stools, this difficulty can readily be overcome by boiling the milk. Boiling the milk has a more marked and more certain effect upon the digestibility of casein than such procedures as adding a large amount of alkali or such substances as sodium citrate to the food mixtures. The beneficial effect of boiling the milk can be demonstrated by results seen in cases of dyspepsia and

even in some cases of enteritis if boiled skimmed milk dilutions are used. The curds and mucus of the stool are promptly replaced by a smooth, salve-like residue.

The Fat of Cow's Milk.—The fat of cow's milk differs from that of woman's milk in a higher content of the volatile fatty acids which fact is given as one of the explanations for its being less digestible. While both cow's milk and woman's milk contain approximately the same amount of fat, namely 4 per cent, nevertheless it is a well established clinical fact that the majority of artificially fed infants cannot take this amount of fat over a prolonged period of time. It has been found that dilutions of whole milk furnish not only a sufficient amount but also as much fat as can usually be well borne by the infant. As the infant grows older its tolerance for fat increases, just as is the case with the casein.

Fat overfeeding produces vomiting, diarrhea with fat curds in the stools, or constipation with large, dry, calcium soap stools and eventually metabolic disturbances such as eczema and acidosis. The amount of fat entering into the infant's diet must therefore always be carefully supervised.

The Carbohydrate of Milk.—Cow's milk and woman's milk both contain lactose, or sugar of milk, as their carbohydrate constituent. So far as we know, there is no difference in the digestibility or in their chemical behavior. In artificial feeding, however, we frequently encounter trouble when lactose is used as an addition to the milk; excessive fermentation leading to the development of a fermental diarrhea with excoriating stools; excoriated buttocks, fever and loss of weight often result from its use. It has also been observed that infants do not, as a rule, gain as rapidly when taking milk mixtures containing milk sugar as when other sugars are used. Cane sugar is not fermented into lactic acid in the intestinal tract and frequently agrees better than milk sugar. The popular vogue of condensed milk rests partly upon the fact that it contains cane sugar and that it is readily digested and is fattening. The most fattening

sugar is maltose. Maltose can be fed in larger proportions than sugar of milk and the condition of the bowels can be readily regulated by means of varying the amount of maltose in the food. Maltose is never given in pure form but is combined with dextrine. The dextrine is an advantage since it controls the laxative action of the maltose to some extent and furthermore it makes it possible to keep the malt in the form of a powder which is not possible with the hygroscopic pure maltose. Among the commercial malt preparations available for infant feeding Dextrimaltose, Mellin's Food and Borchardt's malt sugar may be mentioned. Cane sugar is the heaviest of the sugars, two level tablespoonfuls equaling one ounce by weight. Sugar of milk and Dextrimaltose are lighter and require three level tablespoonfuls to make an ounce.

Barley water and other cereal decoctions are frequently used as diluents instead of plain water. Barley-water influences the digestibility of the casein favorably, overcoming the *tendency* to diarrhea which cow's milk sometimes induces. Oatmeal-water is often beneficial in overcoming constipation. Besides these physical effects, the cereal decoctions possess slight food value.

The Quantity of Food Required.—The gastric capacity is about one ounce at birth and it increases at the rate of an ounce a month. During the first two months, therefore, the infant should receive from two to three ounces at a feeding. At three months it should receive four ounces and at five months six ounces and so on.

The intervals for feeding should be so regulated that the stomach has had time to empty itself before the next meal is taken. This requires about three hours for a weak food and four hours for a stronger formula. Up to five months, therefore, the infant should be fed every three hours and after that time every four hours. The proper dilution of the milk, the amount for each feeding and the interval between feedings may be summed up as follows:

1 mo.	$\frac{1}{3}$ milk,	$\frac{2}{3}$ water,	1 oz. sugar;	2 oz.	3 hours;	8 bottles in 24 hrs.
2 mo.	$\frac{1}{2}$ milk,	$\frac{1}{2}$ water,	$1\frac{1}{2}$ oz. sugar;	3 oz.	3 hours;	7 " " " "
3 mo.	$\frac{1}{2}$ milk,	$\frac{1}{2}$ water,	$1\frac{1}{2}$ oz. sugar;	4 oz.	3 hours;	6 " " " "
4 mo.	$\frac{1}{2}$ milk,	$\frac{1}{2}$ water,	$1\frac{1}{2}$ oz. sugar;	5 oz.	3 hours;	6 " " " "
5 mo.	$\frac{2}{3}$ milk,	$\frac{1}{3}$ water,	$1\frac{1}{2}$ oz. sugar;	6 oz.	3 hours;	5 " " " "
6 mo.	$\frac{2}{3}$ milk,	$\frac{1}{3}$ water,	$1\frac{1}{2}$ oz. sugar;	7 oz.	4 hours;	5 " " " "
7-8 mo.	$\frac{2}{3}$ milk,	$\frac{1}{3}$ water,	$1\frac{1}{2}$ oz. sugar;	8 oz.	4 hours;	5 " " " "
9 mo.	$\frac{3}{4}$ milk,	$\frac{1}{4}$ water,	1 oz. sugar;	9 oz.	4 hours;	5 " " " "
10-11 mo.	$\frac{3}{4}$ milk,	$\frac{1}{4}$ water,	1 oz. sugar;	10 oz.	4 hours;	5 " " " "
12 mo.	whole milk, 10 oz. at a feeding.					

During the first month an infant requires from eight to ten ounces of milk and one ounce of sugar to meet its caloric requirements. From the second to the fourth month it needs about one pint of milk and one and a half ounces of sugar. From the fifth to the seventh month twenty-four ounces of milk and one and a half ounces of sugar will be needed. During the eighth and ninth months it will need twenty-eight ounces of milk and one ounce of sugar. The following table gives the formulæ which we have used for some time at the Mothers' Clinic at the Hahnemann Hospital. Owing to their simplicity and their applicability to the average normal case they can safely be recommended for general use.

1 to 2 mos. . . 3 ounces every 3 hours. . . 7 bottles in 24 hours.

Formula: Milk, 8 oz.; water, 12 oz.; sugar, 1 oz.; or 2 level tablespoons.

3 mos. 4 ounces every 3 hours. . . 7 bottles in 24 hours.

4 mos. 5 ounces every 3 hours. . . 6 bottles in 24 hours.

Formula: Milk, 1 pint; water, 14 oz.; sugar, $1\frac{1}{2}$ oz.; or 3 level tablespoons.

5 mos. 6 ounces every 4 hours. . . 5 bottles in 24 hours.

6 & 7 mos. . . 7 ounces every 4 hours. . . 5 bottles in 24 hours.

Formula: Milk, 24 oz.; barley water, 12 oz.; sugar, $1\frac{1}{2}$ oz.; or 3 level tablespoons.

8 mos. 8 ounces every 4 hours. . . 5 bottles in 24 hours.

9 mos. 9 ounces every 4 hours. . . 5 bottles in 24 hours.

Formula: Milk, 32 oz.; barley water, 12 oz.; sugar, 1 oz.; or 2 level tablespoons.

Top-Milk Dilutions.—Infants that do not gain satisfactorily upon whole milk dilutions are sometimes benefited by a change to top-milk. The advantages of top-milk are its high caloric value and its laxative effect in certain cases of constipation. Before resorting to top-milk, however, we must first satisfy ourselves that the infant is capable of digesting a high-fat formula. If there has been much spitting up or vomiting the additional cream in the formula will most likely aggravate the symptoms. If the infant is suffering with constipation the stool should be examined according to the method previously described. If the constipation is due to the presence of calcium soap in excess in the stool the addition of fat to the formula will only aggravate the condition. This type of constipation calls for a still further reduction in the fat and an increase in the carbohydrate.

Top-milk dilutions are sometimes better tolerated than whole milk mixtures of equal caloric value owing to the fact that the ratio of casein to fat is decidedly lower than in whole milk. Most infants can take a fairly high percentage of either fat or protein provided both of these elements are not present in the food in high percentages at the same time. Most infants can digest undiluted skimmed milk; if, however, the fat is restored to the milk the food becomes too rich for the infant. The same holds good with the protein. When the protein is reduced the fat may be proportionately increased. In whole milk the ratio of fat to protein is 1:1, in a ten per cent top-milk it is 3:1, and in a seven per cent top-milk 2:1. Top-milk must be given in more dilute form than whole milk and so the percentage of protein is low in these formulæ.

One ounce of ten per cent top-milk has a food value of 38 calories. When diluted with two parts water this is reduced to 13 calories. One ounce of whole milk has a food value of 21 calories; the above dilution reduces this to 7 calories. Whole milk formulæ therefore require a much larger proportion of milk than do top-milk formulæ.

If a quart of milk be permitted to stand from four to five hours practically all of the cream will have risen to the top of the bottle at the end of that time. The upper layers of the cream are the richest in fat and if we remove the upper ten ounces from the quart with a Chapin dipper or by carefully decanting the same into a measuring glass we will obtain a mixture of milk and cream containing approximately 10 per cent of fat and 3.5 per cent of protein. This is spoken of as "ten per cent top-milk." By removing the upper sixteen ounces from a quart of set milk we obtain a milk and cream mixture containing approximately 7 per cent fat and 3.5 per cent protein. This is called "seven per cent top-milk."

Ten per cent top-milk is rarely used because of its high fat content and it must be diluted with at least two parts of water before it can be fed to the average baby. A formula of one part ten per cent top-milk, two parts water and five per cent sugar contains approximately $3\frac{1}{3}$ per cent fat, 1.2 per cent protein and 6.5 per cent sugar. This formula corresponds to the percentage composition of woman's milk in the early period of lactation. Unfortunately, however, such a formula will not take the place of breast milk with a young infant with feeble digestion.

Seven per cent top-milk when diluted with two parts water gives a formula containing $2\frac{1}{3}$ per cent fat and 1.2 per cent protein. This amount of fat is more likely to agree with a young or delicate infant and this formula is therefore valuable when a relatively high fat per cent and a relatively low protein per cent are indicated. When diluted with an equal part of water seven per cent top-milk gives a formula containing $3\frac{1}{2}$ per cent fat and $1\frac{3}{4}$ per cent protein. This is a useful formula for an infant from three to six months old with normal digestion or for the correction of simple constipation. The formula closely approximates woman's milk in the latter period of lactation and is a good "imitation" of breast milk. Five per cent of sugar must be added in order to bring the sugar percentage up to seven per cent.

Method of Calculating the Ingredients in the Food.—

Having determined the number of calories required by the infant in twenty-four hours according to the rules given above (see table, page 60), the amount of milk and sugar that will be needed to furnish the specified number of calories in the formula must next be calculated. A good general rule for determining the amount of milk is to allow one and one half ounces per pound of body weight. This will supply the adequate amount of protein to cover the child's nutritional needs (Holt). The remaining calories will be furnished by the sugar entering into the formula. The water in the formula must be sufficient to make up the amount of food required in twenty-four hours. The following example should make these directions clear: An infant four months old weighing 14 pounds and requiring 42 calories per pound will need a formula furnishing approximately 590 calories. If one and one half ounces of milk per pound of body weight are allowed in our calculation then 19 ounces of milk will enter into the formula. This amount of milk will furnish 380 calories (one ounce of milk equals 20 calories). Subtracting 380 from 590, the total number of calories required, we have a deficiency of 210 calories. The latter must be made up by the sugar of the formula. One ounce of sugar has a food value of 120 calories, therefore it will require one and three quarter ounces of sugar to furnish the remaining 210 calories.

Having fixed the amount of food stuffs entering into the formula we must next determine the amount of water. Water must be added in sufficient amount to make the proper quantity of food for twenty-four hours. The infant's age decides this question. At four months the average capacity of the stomach is five ounces; the infant should receive six bottles at three hour intervals. The amount required in twenty-four hours is therefore thirty ounces. Since nineteen ounces of milk enter into the formula a balance of eleven ounces of water is needed to make up the deficit. The formula now reads as follows: 19 ounces milk, 11 ounces water, $1\frac{3}{4}$ ounces sugar. Divide into 6 bottles of 5 ounces each; feed every 3 hours.

Another method recommended is to fix a definite amount of sugar for the formula based upon the infant's age or weight. Clinical experience has taught us that the normal infant from one to two months old, or an infant under ten pounds weight, requires one ounce of sugar in twenty-four hours. From the third to the sixth month it needs one and one half ounces. Perhaps more accurately stated we should allow five per cent of sugar added to the formula; in other words, for a twenty ounce formula, one ounce of sugar; for a thirty ounce formula, one and one half ounces of sugar and for a forty ounce formula, two ounces of sugar. Consequently, if we know the amount of food required in twenty-four hours we can first determine the amount of sugar entering into the formula, compute its caloric value, subtract this from the total number of calories needed and make up the balance with milk, allowing 20 calories per ounce of milk. The result in the above cited case would be slightly different because we would allow only one and one half ounces of sugar (five per cent of thirty ounces) which would furnish 180 calories. In order to supply the remaining 410 calories we would have to use $20\frac{1}{2}$ ounces of milk. Absolute mathematical accuracy in the making of these formulæ is unnecessary, however, and if we gave directions for using 20 ounces of milk, one and one half ounces of sugar and 10 ounces of water we would come near enough to the mark for all clinical purposes.

Method of Estimating Percentages in a Milk Formula.—The percentage of fat and protein in a milk formula can readily be calculated from the number of ounces of milk, cream or top-milk entering into the formula. We must, of course, know the percentage of fat in the milk, cream or top-milk used in the formula. The following rule is a simple method for obtaining the above data: "Multiply the percentage of the ingredients entering into the the formula by the number of ounces of the same used in the formula and divide the product by the total number of ounces in the formula." For example,

given a formula containing 10 ounces of 7 per cent top-milk and 10 ounces of water, the percentage of fat and protein are calculated as follows: 7 per cent top-milk contains 7 per cent fat and 3.4 per cent protein. The percentage of fat in the formula is: 7×10 divided by 20 equals 3.5 per cent. The percentage of protein is: 3.5×10 divided by 20 equals 1.75 per cent.

Method of Estimating the Number of Calories in a Milk Formula.—The caloric value of a food can be estimated directly from the percentage of fat, protein and carbohydrate which it contains, remembering that one gram of fat furnishes 9.3 calories and one gram of protein and one of carbohydrate furnish 4.1 calories respectively. Fraley has devised a simple formula for making these estimations which gives the *caloric value of the food per ounce*. “Multiply the percentage of fat by two, add the sugar percentage and the protein percentage and multiply the result by 1.3.” To illustrate, we will apply this rule in the case of cow’s milk. The food value of cow’s milk is usually given as 21 calories per ounce. Its percentage composition is 4 per cent fat, 3.5 per cent protein and 4.5 per cent sugar. Therefore, $8 \text{ plus } 3.5 \text{ plus } 4.5$ equals 20.8 C.

The most practical method, however, of estimating the food value of a formula is to compute the same directly from the ingredients entering into the formula. Since the food value of an ounce of cow’s milk is 21 calories and that of an ounce of sugar is 120 calories the number of ounces of milk and sugar entering into the formula represents the caloric value of the food. Similarly, if top-milk or cream be used, the calories can be calculated on the same basis (see table, page 50).

The Use of Lime Water and Other Alkalies.—The addition of lime water and other alkalies to the infant’s food is frequently recommended. This procedure, however, is based upon a misconception concerning the digestibility of the casein of cow’s milk and also upon the erroneous belief that while mother’s milk was alkaline in reaction, the milk of the cow was

acid in reaction and should therefore be rendered alkaline. Theoretically, the coagulation of cow's milk in the infant's stomach can be delayed or even prevented by the use of alkalis. This, however, is not a desirable result to be obtained. The acidity of the gastric contents is a prerequisite to normal digestion (see ante). Furthermore, should the casein of the cow's milk cause digestive disturbances these can be overcome much more satisfactorily by boiling the milk than by the addition of alkalis to the food. The use of lime water for the purpose of adding calcium to the food is illogical because the casein of cow's milk contains more than enough calcium to supply the metabolic needs of the infant.

Pasteurization: Boiled Milk.—The question whether milk should be fed in its raw state or whether it should be sterilized by means of pasteurization or boiling depends upon circumstances. Naturally it would seem most logical to employ raw milk, with its vitamins and ferments intact, rather than give the infant a “dead,” sterilized food. Raw milk, however, is a dangerous bacteria carrier and it is therefore safe to use it in the raw state only if it is known to come from absolutely healthy cows and milked and bottled under the strictest sanitary precautions. These requirements are fulfilled in the case of “certified” milk and such milk is safe in so far as its bacteriological standing is concerned. Raw milk, however, is at times not well digested and in such cases it should be boiled for the purpose of changing the physical character of the casein. Raw milk forms a more or less tough, firm curd in the stomach while boiled milk yields a softer, more digestible curd. Boiling the food, therefore, must be considered from the standpoint of its effect upon the digestion as well as a safe and simple method of sterilization.

Pasteurization does not alter the taste nor the digestibility of the milk as does boiling. Commercially pasteurized milk is not altogether safe from the bacteriological standpoint. All ordinary dairy milk, therefore, should be pasteurized before

being given to the infant. This is best done by pasteurizing the food directly in the bottles after the formula has been made up. The Freeman Pasteurizer is a convenient apparatus for this purpose. If a pasteurizer is not available the filled bottles, stoppered with pledgets of cotton, should be immersed in a pail or dish-pan filled with water up to the level of the food in the bottles. The water is slowly heated to 160 degrees F. and then the pan is taken from the fire and covered with a heavy cloth to prevent too rapid cooling of the water. At the end of thirty minutes the bottles are rapidly cooled in running water and kept on ice.

It has been urged that pasteurized milk has all the advantages of raw milk while there is not the danger of scurvy and rickets developing with its use as is the case when boiled milk is fed over a prolonged period of time. This assertion, however, lacks clinical proof. Scurvy and rickets do not develop solely from boiling the milk. Furthermore, when artificial foods are used the child should early receive orange juice and vegetable broths. When this is carried out there is no danger, per se, from boiled milk. Boiling the milk is a simple and surer method of sterilization than pasteurization.

The Preparation of the Food.—The utensils that will be required for the preparation of the infant's food are: an agate-ware saucepan holding two quarts, for mixing the food; a double boiler, for preparing barley-water and other similar preparations; a large kitchen spoon for stirring the food; a sixteen ounce glass graduate; a pitcher and a funnel. It is wise to keep these utensils exclusively for the baby's diet kitchen.

Since the total amount of food for the twenty-four hours is made up each morning there should be on hand a sufficient number of bottles and a wire bottle rack so that the proper amount of food can be put into each bottle and the bottles immediately placed on ice. In selecting a bottle the main point to bear in mind is the cleaning of the same; round bottles with a fairly wide neck into which a bottle brush can readily

be introduced are the preferable pattern. An infant under four months old requires a bottle holding six ounces; up to six months an eight ounce bottle and after that time a ten or twelve ounce bottle. If we allow for an empty space of an ounce or two in the bottle the infant will be better able to nurse than if the bottle is filled up to the neck.

The best nipples are those which do not readily collapse and which can be inverted so that they can be thoroughly scrubbed and rinsed.

All instructions for preparing the infant's food should be given in writing. Many serious errors in the preparation of the food are made through a lack of proper instruction or through a misunderstanding on the part of the mother. When raw milk is to be used in the formula it is most important that the sugar be first dissolved in warm water and the solution be allowed to cool before milk is added. The milk should never be permitted to become warm excepting just before feeding the baby when it must be warmed to blood heat by placing the bottle in a saucepan of hot water.

The author makes it a practice of supplying his patients with a feeding-blank, properly filled out, which gives instructions for preparing the formula as well as indicating the proper amount of each ingredient. Patients are instructed to report at regular intervals so that the baby's progress in weight and the state of its digestion can be followed. With each report a specimen of the baby's stool should be brought for inspection and chemical examination if necessary.

All bottle-fed infants should receive orange juice after the fourth month. This may be given in doses of a teaspoonful gradually increased to three or four teaspoonfuls diluted with an equal amount of water. The best time to give the orange juice is one hour before feeding.

Milk	quart, pint, ounces
Top milk (upperoz. from a quart)	ounces
Cream	ounces
Water, barley-water	ounces
Sugar, or food	level tablespoonfuls.

Directions: (a) Boil some water for five minutes, take from fire and measure out the amount specified above. Add the to the water, dissolve thoroughly and let the solution get cold. Then add the milk, which should be ice cold, mix thoroughly by stirring with a large spoon and fill the bottles, pouring ounces into each of bottles. Stopper the bottles with pledgets of sterile cotton and put on ice immediately.

(b) When the food is to be *boiled* prepare as follows:

Mix the milk and water and boil for three minutes, constantly stirring to prevent the formation of a scum. Take from the fire and add the sugar, or food. Fill the bottles, cool rapidly in running water and put on ice.

Feeding Schedule:oz. every ...hours ...A. M. ...P. M.

Always use a fresh, unopened bottle of milk for the baby's formula.

Empty the nursing bottle immediately after each nursing, scrub with a bottle brush, using warm soap-suds, then rinse with plain warm water. Boil the bottles every morning before filling.

The nipples should be rinsed after each feeding, turned inside out to thoroughly cleanse them, boiled for five minutes, rapidly dried and then kept in a sterile fruit jar, or a jelly glass with a tight fitting cover.

Barley water is prepared as follows: Take one level tablespoonful of barley flour and blend into a thin smooth paste with a little cold water. Pour into a pint of boiling water containing a pinch of salt. Boil slowly in an open saucepan for five minutes, stirring occasionally, then transfer to a double boiler, cover and cook for twenty minutes. Strain, and add sufficient water to make one pint.

FEEDING DURING THE SECOND YEAR.

When the infant is a year old it should be able to take milk in its natural state, that is, without the addition of water or sugar. Some infants, however, have an abnormally low tolerance for cow's milk and in such cases it may be necessary to still remove part of the cream or add water to the milk even after they have attained this age. Other foods, notably cereals

and green vegetables, properly prepared, should now form an important part of the dietary and the child should be weaned from its bottle.

Cereals must be thoroughly cooked; a convenient way of preparing them is to let them cook in a fireless cooker over night so that they will be ready early in the morning. The uncooked cereals are not suitable for children.

Vegetables should first be added to the diet in the form of a vegetable soup. This contains the mineral salts and vitamins so essential to the child's normal growth and is also an excellent food to correct constipation. Older children may have any of the tender fresh vegetables, well cooked and mashed. Baked potato and boiled rice may be given at one year of age.

Bread and *Zwieback* can be given as the molar teeth appear. *Butter* is often digested better than cream and may be used on cereals and vegetables when cream disagrees.

Eggs are rich in fat and iron and also contain the fat-soluble vitamin. They are therefore a most important food and should be gradually introduced into the child's diet. They are best given cautiously as many children show evidence of anaphylaxis to egg albumin.

Meat is not required until the latter part of the second year at which time it may be given several times a week. There is no especial preference as to the kind of meat so long as it is fresh and tender. Older children may have meat once a day. For a young child it should be minced or cut very fine.

Desserts. The best desserts for children are the simple milk puddings and custard. The latter may be used for introducing eggs into the child's diet and in place of a meat course in the meal. Gelatine, on account of its high lime content, may occasionally be given. *Fruit* is best given stewed and makes an excellent dessert especially in conjunction with a meat menu. Bananas have a high food value but their starch content makes them rather indigestible; they are therefore best given baked. A banana can be baked like a sweet potato, in from ten to

fifteen minutes. Peaches, if thoroughly ripe, may usually be given raw (peeled).

The following diet lists will be found applicable for the average normal child of the ages specified.

DIET FOR A CHILD 1 YEAR TO 18 MONTHS

- 7 A. M. Half a teacup of cream of wheat or strained oatmeal with two ounces of warm milk and a teaspoonful of sugar; six ounces of warm milk.
- 9 A. M. Juice of half an orange.
- 11 A. M. Ten ounces of warm milk from a bottle. Sleep until 1.30 P. M.
- 2 P. M. (a) A cup of strained vegetable soup; a small baked potato; cup custard or junket; a slice of stale bread with butter or home-made fruit jelly, or
 - (b) One ounce of beef juice poured over a slice of bread or mixed with half a small baked potato; a tablespoonful of strained green vegetable (spinach, string beans, peas, carrots, asparagus tips, stewed celery); the pulp of three or four stewed prunes, or a small baked apple, or
 - (c) A cup of cream soup, or broth with rice, a tablespoonful of green vegetable, stewed fruit as above, a thin slice of stale bread with butter.
- 6 P. M. Boiled rice or a cooked cereal with warm milk, the same quantities as at 7 A. M., six ounces of warm milk from a cup.

DIET FOR A CHILD 18 MONTHS TO 2 YEARS

- 7 A. M. A teacupful of cooked cereal with warm milk and a teaspoonful of sugar (oatmeal, cream of wheat, wheatena, wheaten grits, Pettijohn's cream of barley); one egg, soft boiled or coddled; bread and butter; a cup of warm milk.
- 11 A. M. Eight ounces of warm milk from a cup. Sleep until 1.30 P. M.
- 2 P. M. Give the following combinations on successive days:
 - (a) Eight ounces of strained vegetable soup; a tablespoonful of minced white meat of chicken; bread and butter or bread and jelly; junket or gelatine.
 - (b) Two to three tablespoonfuls of a strained green vegetable, such as spinach, carrots, squash, peas, string beans, tender lima beans, asparagus tips, stewed celery; two tablespoonfuls of a starchy vegetable, as rice or macaroni or mashed

potato; a tablespoonfull of minced lamb; four ounces apple tapioca pudding or a baked apple.

- (c) A medium sized baked potato with butter or beef juice; a green vegetable as above; a cup of warm milk; bread and butter; a cup custard or bread pudding.

6 P. M. A teacupful boiled rice or a cereal with warm milk or milk toast; a cup of milk or cocoa; bread and butter; fruit jelly or jam or stewed fruit.

DIET FOR A CHILD 3 YEARS AND OVER.

There should be three meals a day. A child of three years of age requires about 1500 calories in 24 hours. The table given on page 51 will be found helpful in computing the caloric value of the foods commonly entering into the child's diet. A glass of milk, or milk and crackers, may occasionally be given between meals in the case of very active children who are always hungry or in cases of children with poor appetites who, under no circumstances, will eat a sufficient amount of solid food at their meals.

Breakfast should consist of a cooked cereal, a soft-boiled or poached egg, bread and butter and a cup of warm milk.

Dinner may be arranged according to the schedule for a two year old child excepting that the quantities must be larger and there is no necessity for straining the vegetables. It is better to give the child its heaviest meal in the middle of the day and give a light *supper* consisting of a cooked cereal or rice; bread and butter, a cup of cocoa and some stewed fruit. Three to four glasses of milk should be taken daily as this is still the most important food for the growing child. When, however, a child refuses to eat sufficient solid food it is necessary to reduce the milk in the diet until this error is corrected.

Forbidden Foods.—*Candy* usually heads the list of forbidden articles of diet. Children are better off without candy and should not be encouraged to develop the candy-eating habit. However, the craving for sweets is a perfectly natural one in the growing child which requires a high-caloric diet and sugar furnishes a food of high caloric value. A piece of pure candy

after a meal can do no harm. The craving for candy can largely be overcome by giving the child plenty of fruit and a sufficient amount of sugar in its diet (on cereals and in desserts).

Meat.—All fried meats; dried beef; kidneys; pork (excepting bacon); sausage; duck and goose; warmed-over meats.

Vegetables.—Fried vegetables; cabbage; green corn; cucumbers and pickles; all raw vegetables.

Bread and Cake.—Hot bread; griddle cakes; heavy cakes like chocolate cake and fruit cake.

Desserts.—Store candy; nuts; pie; raw fruit and bananas excepting as above.

SPECIAL FOODS AND PROPRIETARY FOODS.

Barley-Water is a valuable food for temporary use during acute illnesses especially when diarrhea is present. It is also used frequently as a diluent for cow's milk, and is the best form of starch to introduce into the infant's diet. Barley-water may be prepared from the grain or more quickly from the flour.

When made from the grain two tablespoonfuls of barley, previously washed, are cooked with a pint of water and a pinch of salt for two hours and then strained. Water should be added to keep up to one pint.

To make barley water from the flour take one level tablespoonful of barley-flour and blend into a thin paste with a little cold water. Pour into a pint of boiling water containing a pinch of salt. Boil slowly in an open saucepan for five minutes, stirring occasionally, then transfer to a double boiler, cover, and cook twenty minutes. Strain, and add sufficient water to make one pint.

Albumin Water.—This is a useful preparation in cases of vomiting. It is made by taking the white of one egg, placing the same in a saucer and cutting it up with a clean pair of scissors, then stirring it into a glass of ice cold water, mix thoroughly and strain. A pinch of salt and a little sugar may usually be added.

Whey.—Take one quart of milk, warm to 100° F. to 105° F. and add one tablespoonful essence of pepsin. Let it stand half an hour, then pour into a cheese-cloth bag and let the whey drip off.

Beef-Juice.—The most palatable preparation is made by slightly boiling a piece of lean beef and then squeezing out the juice with a meat press or a potato ricer. It should be served warm with a pinch of salt or poured over bread or baked potato.

Vegetable Soup.—Take one pound of meat (knuckle of veal or neck of lamb), let it stand in one quart of cold water 1 hour. Bring to boiling point slowly and let simmer one hour. Let stand until cold and remove all particles of fat. Then add one medium sized potato, diced; one carrot, diced; one tablespoonful of rice, one slice of onion and a teaspoonful of salt. Cook slowly one hour. Mash through colander.

Albumin Milk.—Take one quart of whole milk, heat to 100° F. and add two teaspoonfuls essence of pepsin or a junket tablet previously dissolved in a little water. Let it stand one hour, then hang in a cheese-cloth bag for one hour to drain off the whey. Place the curd in a fine wire sieve and rub through with a pint of buttermilk, repeating this process three times. The resulting product is a thin, gruel-like mixture of finely divided curds suspended in the buttermilk. It should be kept in a glass jar on ice and thoroughly stirred before using. It should be fed lukewarm; heat causes it to coagulate.

Keller's Malt Soup.—Dissolve 2½ ounces by measure or 4 tablespoonfuls of Malt Soup Extract in 22 ounces of warm water.

Dissolve 2⅔ ounces by measure or 5½ level tablespoonfuls of wheat flour in 11 ounces of milk and strain through a sieve.

Mix all together and bring slowly to a boil over a slow fire or in a double boiler, stirring repeatedly, and boil 5 minutes.

Proprietary Foods.—A number of artificial infant foods are proprietary preparations which are largely advertised, some to laity, others to the profession exclusively. Many of these are

of practical use to the physician because they meet the demands for certain food-stuffs which can be employed advantageously in infant feeding. There is no doubt that much harm is frequently done by the ill advised use of proprietary foods especially when they are given over a long period of time and without professional supervision. It is therefore important for the physician to know the composition of the various artificial foods on the market in order that he may judge their merits or shortcomings without prejudice.

Proprietary foods may be classified as those which serve merely as milk modifiers, supplying the deficiency of carbohydrate of cow's milk, e. g., Mead's Dextri-Maltose; Mellin's Food; or those which accomplish a partial digestion of the milk through the presence of a digestive ferment (Peptogenic Powder; Benger's Food). Again others are complete foods, requiring only the addition of water in their preparation (Nestle's Food; Condensed Milk). The latter are useful in cases of emergency or when fresh milk cannot be obtained; they should never be used over a prolonged period.

The following list gives a description of the better known proprietary foods together with their percentage composition.

Robinson's Patent Barley-Flour; Brooks' Baby Barley.—Barley flour contains about 8 per cent protein. It is used for preparing barley-water which enters so largely into the composition of modified milk formulæ. Barley-water as ordinarily prepared contains about 1 per cent starch and has a food value of approximately 2 calories per ounce.

Mead's Dextri-Maltose.—This is a product of dextrin and maltose in about equal parts, resulting from the action of diastase upon the starch. It is a pure soluble carbohydrate intended solely to supplement the carbohydrate deficiency of cow's milk. Dextri-Maltose is put up in two forms, No. 1 and No. 2, the former contains salt which is a desirable addition to most milk dilutions. According to the manufacturers the composition of Dextri-Maltose No. 1 is 51 per cent maltose, 42 per

cent dextrin, 2 per cent sodium chlorid. The food value is given as 120 calories per ounce. It is manufactured by Mead Johnson & Co.

Mellin's Food.—Mellin's food is a maltose and dextrin preparation made from wheat and malted barley by the Leibig process. It is freely soluble in water and contains approximately 59 per cent maltose, 21 per cent dextrin, 10 per cent protein and 2.5 per cent potassium carbonate artificially added. The caloric value is given as 105 calories per ounce.

Eskay's Food.—This food is composed of a mixture of barley, wheat and oats, thoroughly baked, with the addition of 54 per cent sugar of milk and some whole egg. The food is prepared by cooking the same with water and adding the decoction thus derived to fresh milk. The caloric value is given as 120 calories per ounce, dry. It is manufactured by Smith, Kline & French Co.

Nestle's Food.—This food consists of a mixture of cow's milk evaporated to dryness with cane sugar and ground wheaten biscuit. It is prepared by the addition of water only. It is poor in fat and contains a total of 74 per cent carbohydrates.

Horlick's Malted Milk.—Malted milk is a preparation containing whole milk and the extracts of malted barley and wheat evaporated to dryness and converted into a powder. It is a complete food requiring the addition of water only. Horlick's Malted Milk contains 8.78 per cent fat, 16.35 per cent protein, 18.8 per cent dextrin, 10.65 per cent lactose, 38.5 per cent maltose, 3.86 per cent salts. The caloric value is given as 121 calories per ounce.

Condensed Milk.—Eagle Brand condensed milk, made by Borden's Condensed Milk Co., consists of cow's milk evaporated at a temperature of 212 degrees Fahrenheit to about one quarter its original bulk with the addition of cane sugar. A number of other similar preparations are on the market. Its composition is given by the manufacturer as follows: 9.5 per cent

fat, 7.84 per cent protein, 53.67 per cent sugar. Its food value is 130.6 calories per ounce.

One part of condensed milk to eight parts of water gives a mixture containing 1.33 per cent fat, 1.09 per cent protein and 7.48 per cent carbohydrates. The objection to such a formula is the high carbohydrate and the low fat and protein content.

Unsweetened Condensed Milk is prepared from whole milk without the addition of sugar. It is not as concentrated as the sweetened variety and requires less dilution. Sugar or malt preparations may be added to the dilutions as in the case of fresh milk.

Dry Milk Preparations.—Recently dried milk has been commercialized and certain preparations have been especially advocated as infant foods. *Mammala* is sold as an infant food, being a mixture of dry milk and milk sugar (total amount of sugar of milk, 54 per cent). *Dryco Brand* of dry milk is made from milk from which part of the fat has been removed but no sugar has been added. Dry milk often agrees with infants who cannot take plain milk and is an excellent food to use in traveling and whenever it is impossible to obtain good, fresh milk. When one part by weight of Dryco is mixed with eight parts water (one level tablespoonful to one ounce of water), the following strength of food is obtained, 4 per cent protein, 1.5 per cent fat, 5.5 per cent sugar of milk. One level tablespoonful of Dryco has a food value of 16 calories.

Peptogenic Powder, manufactured by Fairchild Bros. and Foster, is a mixture of sugar of milk with pancreatic extract and bicarbonate of soda. By adding this powder to milk dilutions and warming the same to blood heat for 8 minutes the casein of the milk is partially predigested and the deficiency in carbohydrates of the cow's milk is overcome. *Benger's Food* is a farinaceous food containing pancreatic extract. It is prepared by mixing the food with a little cold milk, then adding the balance of the milk and water, boiling hot, and allowing

the mixture to stand for 15 minutes. During this time the starch and casein are partially digested. Predigested foods are useful in acute digestive disturbances or for temporary use in cases of feeble digestion but they should not be used over a prolonged period.

Malt Soup Extracts.—*Borcherdt's* Malt Soup-Extract and Maltine Malt Soup-Extract are preparations of malt syrup to which potassium carbonate has been added. They are used in the preparation of Keller's Malt Soup which is an excellent food in cases of obstinate constipation and in malnutrition and marasmus resulting from overfeeding. The formula made according to Keller contains low fat and protein and high carbohydrate percentages.

CHAPTER V.

DISEASES OF THE NEWBORN.

CONGENITAL DEFECTS.

Congenital heart disease, congenital defects of the brain resulting in idiocy, and hypertrophic pyloric stenosis are the developmental defects of especial interest to the pediatricist. Their discussion will be found in the chapters dealing with the diseases of those organs. Other defects encountered in the newborn are mainly of surgical and obstetrical interest. *Asphyxia neonatorum* and still-birth frequently result from some congenital malformation and should therefore always be suspected in such cases.

ASPHYXIA.

Asphyxia in the newborn may be of intra- or extra-uterine origin. *Intra-uterine asphyxia* results from the interruption of the placental circulation through compression of the cord or premature separation of the placenta. Respiratory efforts are excited in the child through the resulting carbonization of the blood and the lungs consequently become filled with amniotic fluid.

Extra-uterine asphyxia occurs at the time of or shortly after birth. The degree of asphyxia may be of different grades, varying from a simple interference with the respiratory function from the aspiration of mucus or amniotic fluid into the upper respiratory tract to a complete cessation of respiration. In the latter case the child may be robust when born and present all of the signs of active asphyxia, the body surface being cyanotic and the face bloated (sthenic asphyxia); or it may be pallid and limp and apparently lifeless (asthenic asphyxia). A frequent cause of the asthenic form is injury to the brain

occurring at the time of delivery. Head injuries from protracted labor or forceps delivery usually result in hemorrhage into the pia mater and as a consequence of such a lesion the respiratory centers fail to functionate. In the absence of hemorrhage, malformations of the respiratory or circulatory organs, pulmonary atelectasis, pulmonary syphilis, and premature birth may be mentioned as causes.

The reflexes are not abolished in the sthenic variety and the pulse is slow but perceptible. The vessels of the cord remain full and firm and the infant's muscle tone remains good. The symptoms disappear as soon as respiration is established. It presents a better prognosis than the asthenic variety, in which there is pallor of the body surface, abolition of reflexes, and imperceptible pulse.

Treatment consists in promptly cleaning out the mouth and pharynx and stimulating the respiratory reflex by spanking, the alternate application of cold and warm water and resorting to artificial respiration if necessary. In the asthenic variety the warm bath alone should be employed, together with artificial respiration, but when the asphyxia is symptomatic of one of the serious conditions above enumerated, the prognosis is grave.

CEPHALHEMATOMA.

A cephalematoma is a tumefaction situated upon one of the cranial bones, usually over the parietal, caused by hemorrhage beneath the periosteum. It results from injury sustained during parturition, and is frequently encountered in children born through a narrow pelvis. Being entirely external no pressure symptoms result from such a hemorrhage. The swelling increases in size slightly during the first week and then slowly absorbs. The blood does not coagulate but bone cells are deposited in the periosteum over the swelling so that in cases that are several weeks old a parchment-like feel is imparted to the same. Surgical interference should not be resorted to unless infection occurs.

HEMATOMA OF THE STERNO-MASTOID MUSCLE.

This usually affects the belly of the right sterno-mastoid muscle, most commonly occurring in breach labors, being the result of twisting of the head during parturition. A firm, elastic, egg-shaped swelling appears in the middle of the muscle about two weeks after birth and is accompanied by torticollis. It should disappear in the course of several weeks but sometimes the clot becomes organized and the torticollis persists for a long time, in which case it may require surgical treatment.

INTRACRANIAL HEMORRHAGES.

Apoplexy of the newborn occurs as a venous or capillary hemorrhage of the meninges of the brain, less frequently taking place in the cortex. It results from direct injury during birth. This condition is fully discussed under cerebral palsies. Other forms of injury to the nervous system encountered at this period are facial and brachial paralysis, resulting from pressure or traction upon the nerve trunks supplying these parts.

SEPTIC AND OTHER INFECTIONS IN THE NEW-BORN.

The newborn infant exhibits an apparent immunity to certain of the acute infectious diseases while toward others it shows the same susceptibility as an older infant. No doubt some of the immunity depends upon the presence of antibodies in the system derived directly from the mother through the placental circulation. Some of these antibodies may also be present in the mother's milk and for this reason a nursing infant is less susceptible to infections than a bottle-fed infant.

The newborn is, however, highly susceptible to septic infection and the greatest care must be exercised to protect it from any possible septic source. The organisms mainly responsible for the septic manifestations encountered in the newborn are the streptococcus pyogenes and the staphylococcus pyogenes aureus.

The general symptoms associated with sepsis are apathy and

weakness; feeble cry; fever; vomiting and diarrhea; distended abdomen; rapid respirations and cyanosis; at times convulsions. Jaundice frequently develops. The fever sets in toward the latter part of the first week or during the second week and does not yield to the usual therapeutic measures efficacious in gastrointestinal disturbances.

Local manifestations may be demonstrable such as a mucopurulent discharge from the nose; stomatitis; redness of the skin about the umbilicus; protrusion of the umbilicus and dilatation of the epigastric veins; bronchopneumonia and arthritis.

Continued fever in the newborn should always be looked upon as a serious symptom. Rapid respirations and cyanosis with febrile manifestations and gastrointestinal disturbances are much more likely to signify pneumonia than *pulmonary atelectasis*.

Inanition fever develops during the first three or four days after birth and disappears as soon as sufficient water and food are administered.

Intestinal toxemia presents symptoms of fever and gastrointestinal disturbances which, however, promptly clear up after a dose of castor oil (Morse).

A high leucocyte count can usually be demonstrated to confirm the diagnosis of sepsis.

Other clinical types of infectious conditions encountered in the newborn are erysipelas, tetanus and gonorrheal infection of the eyes and joints.

ERYSIPELAS.

This is a form of cellulitis due to local infection with the streptococcus pyogenes. It most frequently originates at the site of the umbilical cord although an abrasion of the skin in any other part of the body may be the starting-point for the disease. The prognosis is usually grave, especially in cases resulting from infection of the umbilicus.

TETANUS.

The bacillus of tetanus may be inoculated at the site of an abrasion of the skin or of a mucous membrane, or it may gain entrance through the umbilical stump.

The symptoms are identical with those observed in the adult, the earliest manifestations being rigidity of the jaws, occurring, as a rule, shortly after the cord has dropped off. The trismus is followed by tonic spasms of the muscles of the neck and extremities, occurring paroxysmally. As a rule, it terminates fatally within a few days, although it may pursue a protracted course and result in recovery. Tetanus *antitoxin* should be administered as soon as the condition is suspected.

GONORRHEA.

Gonorrheal infection of the eyes is the most common form of infection with the gonococcus of Neisser encountered in infancy. Many cases of vulvitis in infants, however, are gonorrheal in nature. In recent years attention has also been called to the fact that arthritis in infants is frequently of gonorrheal origin. The arthritis is multiple in character and is a manifestation of a general septic infection with the gonococcus. Kimball reported eight cases of gonorrheal pyemia in infants in 1903 coming under his charge at the Babies' Hospital in New York. They all terminated fatally.

OPHTHALMIA NEONATORUM.

The gonococcus of Neisser is responsible for the virulent type of conjunctivitis occurring in the newborn which at times results in destruction of the entire eye. When the infant is infected during parturition the symptoms make their appearance on the third or fourth day. In the cases where the symptoms are of later occurrence infection probably took place after birth.

The first signs of the infection are redness and swelling of the palpebral and ocular conjunctiva, puffiness of the eyelids

and catarrhal secretion. The secretion rapidly becomes purulent and the eye-lids infiltrated and leathery. In the virulent cases chemosis is pronounced and the cornea is deprived of its nutrition through compression of the blood vessels at the sclero-corneal margin. The cornea becomes opaque, its epithelium is shed and perforation may occur.

A benign, non-gonorrheal type of ophthalmia is at times seen due to the ordinary pyogenic organisms. It is recognized by its mild course and the absence of the gonococcus in the secretion.

The prognosis should always be guarded; it is especially unfavorable in cases which have not had the benefit of early treatment. The claim is made that from 25 to 30 per cent of all cases of blindness can be blamed on ophthalmia neonatorum.

Treatment.—When the first symptoms of an ophthalmia are observed the eyes should be flushed hourly with a 2 per cent solution of boric acid and kept covered with compresses of an ice cold solution of the same. As soon as the discharge becomes thick and creamy, a few drops of a solution of nitrate of silver, three to four grains to the ounce should be instilled into each eye three times daily. If the discharge continues to increase it is better to discontinue the compresses and irrigate the eyes with several ounces of a warm boric acid solution every half hour. As the gonococci decrease in the pus the nitrate of silver should be used less frequently. As the inflammation subsides and the eye-lids lose their infiltrated character a few drops of a stronger solution of nitrate of silver (2 to 4 per cent) may be dropped upon the everted surface of the lids, care being taken not to permit the solution to get into the eye. This process should be followed by an irrigation with normal salt solution. In order to inspect the cornea satisfactorily from day to day and properly flush out the conjunctival sacs retractors must be made use of.

When the cornea becomes involved a drop of one per cent solution of atropine should be instilled into the eye twice daily. In threatening perforation, esserine should be used. *Aconite*

may be administered internally in the early stages and *euphrasia* in the later stages. The responsibility which these cases involve makes it advisable to always call an oculist in consultation.

ACUTE FATTY DEGENERATION, OR BUHL'S DISEASE.

This disease was first described by Buhl in 1860, and presents parenchymatous inflammation, fatty degeneration and hemorrhages in the heart, liver and lungs. It is probably of infectious origin. It is rare, and is only seen in lying-in hospitals. The children are usually born asphyxiated, and they do not entirely recover from this state. Cyanosis supervenes, and they either die at this time, or the course of the disease is protracted, and bloody diarrhea, hemorrhage from the navel, mouth, nose and conjunctiva, and icterus, set in. Later, edema of the skin occurs, and death from collapse follows at about the end of the second week. The diagnosis can only be positively made by a microscopic examination of the organs. The course is always fatal.

ACUTE HEMOGLOBINURIA, OR WINKEL'S DISEASE.

In 1879 Winkel encountered a series of twenty-three cases of hemoglobinuria occurring in the new-born, associated with cyanosis, icterus, and hemorrhages in the various organs, with a fatal termination within thirty-two hours in the average of cases. The cause is unknown, but it is undoubtedly an infection. Other cases have been reported, but not in such an extensive epidemic as the above. Hamill and Nicholson in a series of carefully studied infections in the new-born ("Archives of Pediatrics," Sept., 1903) found that a variety of microorganisms is to be encountered, showing that careless nursing is responsible for these infections.

MASTITIS.

Inflammation of the mammae with abscess formation may result from attempts to squeeze the milk-like secretion from the

breasts which is normally present in a large percentage of infants. There is no reason for interfering with this secretion which eventually disappears of its own accord. Should an actual mastitis develop hot fomentations and *belladonna* should be prescribed.

VAGINAL HEMORRHAGE.

A bloody vaginal discharge appearing on the fifth or sixth day is occasionally observed in female infants. This usually subsides in two or three days and is of no pathological significance. Vaginal hemorrhage may, however, be a symptom of sepsis. In such cases the bleeding occurs later and is accompanied by other manifestations of sepsis.

ICTERUS NEONATORUM.

Icterus, or jaundice, occurs as a symptom in Buhl's disease, Winkel's disease, in septic infection and in cases of congenital malformation of the bile ducts.

Physiological jaundice of the newborn occurs in about two thirds of all healthy infants, the percentage being somewhat higher in the premature and undersized.

Endless theories concerning the etiology of icterus neonatorum have been advanced. According to Quincke, it results from resorption of bile from the intestine as a result of patency of the ductus venosus Arantii; according to Birch-Hirschfeld it is due to a swelling of the capsule of Glisson following interruption of the circulation in the umbilical vein. Hofmeier considers it of hematogenous origin depending upon an extensive destruction of red blood corpuscles. This process takes place in the liver shortly after birth. Czerny believes that it is due to infection.

The most striking feature associated with a study of jaundice in the newborn is the strong disposition of the infant to develop this condition and the fact that its course is so benign and uneventful. Von Reuss sums up the factors which all seem to contribute to this unusual disposition as follows: (a) Certain

mechanical factors, partly anatomic, partly depending upon the character of the bile itself at this age. (b) The abnormal amount of coloring matter found in the bile and the excessive production of bile pigments in the liver at this age. (c) The functional deficiency and vulnerability of the hepatic cells of the newborn.

The discoloration of the skin is noticed on the second or third day, gradually increases in intensity for several days and then subsides. The jaundice usually clears up during the second week. In delicate and premature infants it may persist for several weeks. Jaundice which does not clear up in a week or two should be looked upon as pathological and not considered in the light of the "physiological" variety.

The stools remain normal in appearance and bile continues to be secreted into the intestine. The urine is not discolored, the bile pigments not being excreted in the urine in solution but they can be demonstrated in the urinary sediment in an amorphous form.

HEMORRHAGIC DISEASE OF THE NEWBORN.

The newborn infant presents a predisposition to hemorrhage owing to the fact that the blood at this time of life fails to coagulate as readily as in the more mature infant. Slight injuries or abrasions are therefore likely to result in a considerable loss of blood with serious consequences. Infections also increase this hemorrhagic tendency and so it is common to find a disposition to hemorrhage as one of the manifestations of sepsis and of syphilis in the newborn.

The characteristic form of hemorrhagic disease of the newborn, however, known as *melenae neonatorum*, occurs independently of traumatism or of an infection, the bleeding taking place spontaneously from the mucous membranes or under the skin. The hemorrhage is usually extensive, is not influenced by the ordinary styptic measures and frequently terminates fatally.

As a rule no lesions are found to account for the bleeding although in a certain percentage of the cases small, round, superficial ulcers can be demonstrated in the stomach and duodenum.

The infant is usually a healthy, well-nourished babe at birth and shows nothing abnormal. Several days after birth blood will be discovered in the stool or the infant vomits bloody gastric contents and a rapidly developing anemia results. If an infant vomits a dark or brownish substance or if the stool contains particles resembling meconium after the third day a test for occult blood should at once be made. The early resort to the subcutaneous or intramuscular injection of human blood serum, from twenty to thirty cubic centimeters, taken from one of the parents, offers the best chances for arresting the condition. When the loss of blood has been large the introduction of a larger amount of citrated blood, through the anterior fontanel, will be necessary.

SUDDEN DEATH IN INFANTS.

Sudden death in the newborn is most frequently due to cerebral hemorrhage resulting from compression of the head during birth or from hemorrhage into the internal organs. The latter occurs most frequently in breach cases and in precipitate labors of large infants.

Malformations of the viscera are a common cause of sudden death in young infants. *Enlargement of the thymus gland* may be a cause of sudden death. While Paltauf denies that pressure from the thymus plays a role in the death of these infants, attributing it to the clinical entity which he has termed *status lymphaticus*, or *lymphatism*, still the theory that thymic death can occur has many adherents, notably in Jacobi. The latter writes: "It (the *thymus* gland) is largest, normally, from the third to the twentieth month; about the ninth month it was found, in usual instances, from 1.5 to 2 centimeters in thickness. As the distance between the manubrium sterni and the vertebral column is but two centimeters about the eighth month of life

the slightest increase of an enlarged thymus through distended circulation, by crying or otherwise, may prove fatal; for besides the thymus, the esophagus, the trachea, the blood vessels, and the sympathetic and pneumogastric nerves are located in that narrow space. Bending the head backward during tracheotomy proved fatal. Swelling of the thymus in a cold bath may be dangerous" (*Therapeutics of Infancy and Childhood*). In discussing a case recently reported by Caille, Jacobi called attention to the fact that but a few are on record since Kopp reported his first case of thymic asthma nearly a hundred years ago. He related a case operated upon by König in which the gland was partly excised with life-saving results. For detailed report on this subject the reader is referred to Jacobi's monograph (*Trans. Ass. of American Phys., Vol. III*).

Atelectasis.—This is either congenital or acquired. Complete atelectasis is seen in *asphyxia neonatorum*. In feeble infants atelectasis may develop after the lungs have been functioning, and if progressive it results in death. It is due to an inability of the infant to adjust itself to its new environment. As long as it received its sustenance and its oxygen from the maternal blood supply it developed normally but when cut loose from this parasitic life it succumbs. Areas of atelectasis develop in the lungs during an attack of bronchitis or bronchopneumonia and may be of sufficient extent to seriously interfere with respiration. In many cases of congenital debility and marasmus the only lesion found post-mortem is pulmonary atelectasis.

Asphyxia from the aspiration of liquids into the larynx is at times a cause of sudden death in feeble infants. Sudden death may arise from *laryngismus stridulus* or during general convulsions. Sudden death with *hyperpyrexia* may occur in almost any of the acute infectious diseases.

CHAPTER VI.

DISEASES OF THE MOUTH.

DENTITION.

The teeth which are erupted during infancy are spoken of as the temporary or milk teeth. They are twenty in number and are gradually replaced by the permanent teeth which number thirty-two. At the time of birth the teeth are present in the alveolar process of the jaw, in an immature form, enclosed in the so-called dental sacs. When the infant is being properly nourished and its mineral salt metabolism is normal the development of the teeth from these tooth germs progresses hand in hand with the development of the jaw and the osseous system. The eruption of the teeth begins at about the sixth month and is completed at the end of the second year. Any condition, however, interfering with the child's nutrition or any constitutional disease which disturbs the calcium metabolism exerts a marked influence upon the development and eruption of the teeth. The result of such pathological conditions is *delayed dentition* and the eruption of imperfectly developed, perishable teeth. The usual order of the eruption of the teeth is as follows:

Six to eight months after birth the two lower central incisors should make their appearance; the upper central incisors usually appear a month later. The upper lateral incisors are the next in order, and at the end of a year the upper anterior molars begin to erupt. At the fourteenth month the lower lateral incisors erupt, followed by the lower anterior molars.

The canine teeth appear between the sixteenth and twentieth months, and at the end of the second year the posterior molars are added to complete the set.

Ordinarily it may be stated that at one year of age there should be six teeth; at one and one-half years of age twelve

teeth; at two years, sixteen teeth and at two and one-half years, twenty teeth.

Soon after the eruption of the milk teeth absorption begins, commencing at the apex of the root and extending to the crown, so that they are either lost by an accidental tearing of the membranous attachment to the gums, or are displaced by the advancing permanent teeth.

The eruption of the permanent teeth begins at the 6th year, the first to come being the molars, or the "six-year molars." These are followed by the incisors, (7 to 8 years). Next to appear are the bicuspid, (9 to 10 years); then the canines at 12 years; the second molars at 12 to 15 years and finally the third molars or "wisdom-teeth" at 17 to 21 years.

Dentition is a purely physiological process, and should therefore run a normal, uneventful course. This is, however, unfortunately not always the case and there is no doubt that many infants are unnaturally peevish and uncomfortable and present digestion derangements when they are teething. Much harm, however, has been done by attributing many disorders to teething which should have been thoroughly investigated and the true cause found and remedied.

As a rule lancing the gums is unnecessary and is to be condemned as a routine practice, but when the gums are tense and irritated and the tooth is ready to come through relief will frequently follow this procedure.

The proper care of the teeth is a hygienic essential as carious teeth are a serious menace to the child's health. A focal infection in the mouth may spread to the tonsils and the lymphatic glands under the jaw and may also induce serious constitutional disturbances. It is also important that the milk teeth be preserved as long as possible, for their premature loss interferes with the growth and development of the jaw, thereby inviting a contracted palate, abnormally small jaw or irregularities in the permanent teeth.

The following remedies are indicated in the disturbances encountered in the teething infant:

Bell. and *Cham.* are perhaps the most frequently employed teething remedies, *Chamomilla* being indicated by irritable temper; greenish, offensive diarrhea, and circumscribed redness of the cheeks. *Belladonna* is indicated when there is fever; flushed face; redness and swelling of the gums; vomiting.

Ferrum phos. is especially useful in anemic infants; cough during teething.

Calcareo phos. is indicated in the delayed teething of rickets and malnutrition. The teeth are imperfectly formed and decay early.

ABNORMALITIES OF THE TEETH.

The most characteristic deformity seen in the teeth is the condition first described by Hutchinson, which occurs in congenital syphilis and is known as "Hutchinson's teeth." The enamel is deficient upon the cutting surface of the upper central incisors and as a result of this defect a semilunar notch is worn into the edge of these teeth. Furthermore, they are shorter than normal and their sides somewhat sloping, giving them the form of a screwdriver, being narrower at their cutting edge than at the root. The canine teeth are also rudimentary and pegshaped.

The milk teeth are not characteristically affected by syphilis; they may be poor in quality and decay early, or they may show irregularities in form and in their enamel covering. Any form of *stomatitis*, however, can affect the development of the teeth.

Rickets delays the eruption of the teeth, and in rachitic children they are, as a rule, abnormally soft and decay early. The permanent teeth may show transverse ridges or a serrated edge as a result of an acute illness occurring during the teething period. Any disease affecting the general nutrition naturally shows its influence upon the teeth if it be active at the time of their eruption.

STOMATITIS.

The term *stomatitis* is applied to the several forms of inflammatory affections involving the mucous membrane of the buccal

cavity. It is a common affection among children, and may be either of mechanical, constitutional, bacterial or myotic origin.

CATARRHAL STOMATITIS.

This form of stomatitis presents an acute diffuse inflammation of the mucous membrane of the mouth.

Etiology.—The exciting cause may be mechanical irritation from a nipple or comforter, or from too vigorous cleansing of the baby's mouth. It may also result from giving the food too hot or from bacterial infection. In the majority of instances, however, catarrhal stomatitis is simply a symptom of some general acute infectious disease or of a gastrointestinal derangement.

Symptoms.—Primarily there is heat and dryness of the mucous membrane of the mouth and gums, together with redness and swelling, which is generally uniform, although it may be more marked in circumscribed areas. This is followed by increased secretion of mucus and saliva, which generally dribbles from the mouth. Pain is present, and the pathognomonic symptom, "The child seizes the nipple eagerly, but after a few seconds drops it with a cry," is explained by this exquisite tenderness of the mouth. The child is fretful and feverish, and, owing to the inability to nurse successfully, soon loses in weight. It is usually of short duration and does not terminate in ulceration.

PITYRIASIS LINGUÆ,

Is a chronic catarrhal inflammation involving the upper surface of the tongue, resulting in the characteristic condition known as *lingua geographica*. It begins as a circular patch or patches of epithelial hyperplasia forming elevated whitish spots, which enlarge and ultimately desquamate in the center, resulting in irregular plaques, with islands of normal mucous membrane interspersed between the hyperplastic epithelium. Several of the ring-like lesions coalesce and form the geographical pattern giving the disease its name. This affection shows great ten-

dency to recur, the interval between the disappearance of the old lesions and the reappearance of a new annular patch being usually of short duration. It is met with in children of all ages, in the healthy as well as sickly, although perhaps most frequently in the rachitic.

APHTHOUS STOMATITIS.

Aphthous stomatitis is characterized by the appearance of small, round, circumscribed, yellowish plaques upon the mucous membrane of the mouth and tongue.

Etiology.—The etiology of this affection is unknown. Forchheimer considered aphthous stomatitis an acute infection of intestinal origin and he compared it to the foot and mouth disease of cattle. Filatow believed it to be a local infection, as it often attacks several children in one family simultaneously. It is most commonly seen from the first to the third year.

Symptoms.—The lesions appear mainly upon the mucous membrane of the anterior portion of the mouth and tongue; they are seldom found upon the tonsils or base of the tongue. They consist of small, round, dirty-white or yellowish plaques, slightly elevated and surrounded by a red areola. There is accompanying pain, salivation, offensive breath, inability to eat, slight fever. The duration is from one to two weeks.

BEDNAR'S APHTHÆ.

This condition is only seen in infants from one to six weeks old; it is characterized by the formation of round, superficial ulcers situated at the bases of the palate. The *prognosis* is usually favorable although deep ulceration of the tissues has been observed. It is no doubt brought on by traumatism from the nipple.

APHTHÆ EPIZOOTICÆ.

This is an infectious form of vesicular stomatitis, resulting from infection with the milk from cows affected with the disease. There is more fever than in aphthous stomatitis, salivation and and coryza are accompanying symptoms, and the vesicles do

not appear on the dorsum of the tongue but are usually situated on the soft palate, lips, gums and cheeks. There is also fetid breath, sometimes vomiting and diarrhea. It runs its course in from one to two weeks.

In *varicella* vesicles often appear in the mouth, but the cutaneous manifestations are sufficient to differentiate it from aphthous stomatitis.

ULCERATIVE STOMATITIS; PUTRID SORE MOUTH.

This variety presents an inflammation of the mucous membrane of the mouth, accompanied by ulceration.

Etiology.—The destructive inflammation of ulcerative stomatitis is due to a local infection. Although it has occurred epidemically, no specific micro-organism has been demonstrated. Smears taken from the lesions show a fusiform bacillus accompanied by a spirillum, identical with the bacteriological findings in *Vincent's angina*. It is only seen in children who have teeth and is often associated with carious teeth.

Pathology.—The process begins with an inflammation of the anterior border of the gums, at the roots of the teeth, most frequently on the lower jaw. Redness and swelling are the initial changes, after which a yellow line, indicating the beginning of the necrotic process, develops along the alveolar border and extends downwards. From the gums the process extends to the inner margin of the lips, and large ulcers are generally formed on the lining of the cheeks opposite to the molar teeth. The sides of the tongue frequently participate, becoming infected by direct contact with the lesions.

Symptoms.—In the beginning of the disease there are the usual symptoms of stomatitis, but soon the characteristic foul breath develops, the pain becomes intense, and prostration and fever is more marked than in the other forms. This is easily understood when we consider the severity of the process and the intoxication resulting from the absorption of the putrid material forming in the mouth. The course is more protracted than

in the other forms of stomatitis and the child's general condition must be improved as well as looking after the local condition.

PARASITIC STOMATITIS; THRUSH.

Parasitic stomatitis is an infection of the mouth due to the development of a parasitic fungus within the mucous membrane. It is characterized by the appearance of milk-white patches which are difficult to remove and have a tendency to coalesce and spread extensively.

Etiology.—The *saccharomyces albicans*, a fungus of the group saccharomyces, is found in the mucous membrane wherever the lesions develop. If a portion of the white pellicle be removed and placed on a slide with a drop of liquor potassæ the mycelium and the spores can be demonstrated.

Artificial feeding, early life, exhausting diseases, catarrhal stomatitis, insufficient salivary secretion, unsanitary surroundings and lack of proper care of the nipples and of the baby's mouth are the etiological factors. The disease can be communicated directly from one patient to another, and is quite common in foundling asylums and among the poorer classes.

Pathology.—The spores of the *saccharomyces albicans*, finding their way into the mouth of the infant, soon develop their mycelia, which penetrate the layers of the mucous membrane and form the white patches or elevations so characteristic of the affection. These patches are difficult to remove, as they are within the mucous membrane, but there is no exudation or pus formation accompanying the process. The lesions usually begin as small, white points on the inner surface of the cheeks, quickly spread and coalesce, so that in a short time the entire buccal cavity and pharynx may be involved. Extension to the esophagus is rare, and to the stomach still rarer, as it confines itself almost exclusively to the squamous epithelium. Rare cases, however, are on record in which these localities were affected, beside the lower rectum the female genitalia, the upper respiratory tract, intestines, and abraded cutaneous surfaces.

Preceding the outbreak of thrush the mucous membrane of the mouth is hot and dry; later there is a sticky mucous secretion, acid in reaction. This is partly due to a lack of the normal alkaline salivary secretion, and to saccharine fermentation, the result of the growth of the fungus.

Symptoms.—Beside the objective symptoms already described there is generally pain due to the catarrhal stomatitis set up by the fungus. The *prognosis* depends upon the infant's condition and as a rule is favorable. In poorly nourished, sickly infants it may be difficult to eradicate.

The white pellicle of thrush closely resembles flakes of coagulated milk and in the beginning is often mistaken for such; but the difficulty with which these spots are removed and the associated stomatitis readily differentiates it from such a condition. Thrush has been mistaken for diphtheritic deposit, but here the age of the patient, together with the associated conditions, the absence of foul breath, glandular involvement, fever and prostration, and the superficial character of the lesions, should readily differentiate the two.

GANGRENOUS STOMATITIS—NOMA.

A destructive inflammatory process involving usually the cheeks and developing secondarily to one of the exanthemata or to some exhausting disease.

Etiology.—It generally follows upon measles, scarlet fever, typhoid fever, or some form of exhausting disease, occurring most frequently between the age of three and six years and in the poorer classes. The pyogenic bacteria, notably the streptococcus pyogenes, are responsible for the destructive pathological changes. In a certain number of cases the diphtheria bacillus has been present (Walsh).

Pathology.—Beginning on the inside of the cheek or near the corner of the mouth, a small vesicle, filled with a turbid fluid, is formed. The vesicle breaks and leaves a superficial ulcer with a hard, infiltrated base, which can be felt through the

cheek. This breaks down and a rapidly spreading gangrenous process develops, with no tendency to limitation. The affected parts become infiltrated and edematous, presenting a shiny, livid appearance.

Symptoms.—Often the first symptom noticed will be the ulcer, as the vesicle is easily overlooked. The breath is foul, prostration profound, and the temperature of the septic fever type. The *prognosis* is unfavorable, the patient either succumbing to septicemia or to a secondary bronchopneumonia; fatal hemorrhage is rare. In the case of recovery there is usually marked deformity.

Treatment of Stomatitis.—All forms of stomatitis can be prevented to a great extent by strict attention to the hygiene of the mouth, as well as by careful supervision of the diet and general hygiene of the child. With artificially-fed babies, it is important to have nipples and bottles kept perfectly clean and sterile. During the course of an acute illness, especially one of the infectious fevers, it is imperative to keep the mouth in a clean condition, for it is in these cases that noma may develop, particularly in the enfeebled and poorly nourished.

Should stomatitis develop, a mild antiseptic wash will be sufficient to carry the case through, excepting in the gangrenous form, which is, strictly speaking, a surgical disease. For this purpose, either a 2 per cent *Boric acid* solution, *alcohol* diluted with three parts water, or, a solution of *potassium chlorate*, one per cent, may be employed. The latter is especially useful in the ulcerative form. In stubborn cases of thrush it may become necessary to touch the patches carefully with a 2 per cent solution of *Silver nitrate*; this is to be followed by rinsing the mouth with salt water.

The *diet* is important in ulcerative stomatitis. By a restriction in the use of all salty articles of food, and the free use of fruit juices and vegetable broths, these cases recover more promptly than under ordinary treatment. Owing to the painful condition of the mouth the diet should be restricted to liquids

and semi-solids, and in older children the use of a tube or feeding-cup with a spout will be very grateful.

Borax is perhaps the most useful remedy in the aphthous and parasitic form, especially in the early stages, with heat and dryness of the mouth. It may be applied directly to the affected parts either in pure form or in the first decimal trituration, which, being slightly sweet, is more pleasant to the child, or it may be used in the form of a saturated solution.

Mercurius may be indicated in all forms, but pathologically it corresponds most closely to the ulcerative form.

Ars.—Thrush; exhausting diseases; prostration; dryness of mouth.

Baptisia.—Ulcerative stomatitis; great fetor of breath; offensive diarrhea; typhoid state.

Bry.—Catarrhal stomatitis; great dryness of mouth.

Hydrastis.—Superficial ulceration; tenacious mucus.

Natr. mur.—Gums spongy; superficial ulcers on tongue and cheeks.

Nitr. ac.—Ulcerative stomatitis; after *mercury*; fetid breath and acrid saliva; acrid diarrhea; cracking of the corners of the mouth.

Rhus tox.—Great restlessness; saliva bloody; lips cracked.

CHAPTER VII.

DISEASES OF THE GASTROINTESTINAL TRACT.

The chief etiological factor in diseases of the stomach in infants is *improper feeding*. This may mean any of the following errors: (a) the use of foods of improper composition; (b) the administration of abnormally large quantities of food; (c) irregularity in the time of feeding, and (d) improper temperature of the food. Beside these factors, infection also plays an important part as a cause of gastric disturbances. Bacteria or their toxins may act either directly upon the mucous membrane of the stomach or toxins circulating in the blood during the course of an infectious disease may induce serious gastric symptoms.

An important fact to bear in mind is the impossibility of drawing a sharp line of demarcation between diseases of the stomach and intestines. In adults there is greater possibility of doing this. The infantile stomach, however, is only a dilation of the alimentary canal and is not completely differentiated from the same. Its position, at first, is almost verticle; its capacity is relatively small and its sphincters are immature. Physiologically it is immature, the main work of digestion falling upon the intestinal tract. Under normal conditions, therefore, the food (breast-milk) is coagulated shortly after reaching the stomach by the rennin of the gastric juice. Hydrochloric acid is now secreted and the casein is converted into acid albumin (syntonin). The action of the pepsin simultaneously secreted is feeble. In fact, the food does not remain long enough in the stomach to be digested completely, the bulk of the digestive process taking place in the small intestine. Vomiting occurs readily and therefore any food that is unsuitable can readily be disposed of and the stomach not

harmed thereby. Should it pass into the intestine, colic and diarrhea usually result.

Vomiting.—Persistent vomiting from birth indicates either cardiac or pyloric obstruction. In the former deglutition sounds are absent, while in the latter the food is generally retained abnormally long before being rejected. At the same time dilatation of the stomach develops together with other signs of pyloric obstruction.

The natural tendency for infants to vomit must not be lost sight of. The cardiac sphincter is poorly developed and owing to the habit of gulping the food too rapidly or overfilling the stomach, vomiting is a common symptom. When the milk is too rich in fat it regurgitates shortly after nursing without being curdled. In indigestion the food is usually vomited an hour or more after nursing and it is curdled and sour. In acute gastritis there is fever; the food is promptly rejected and mucus is present in the vomitus. In pyloric obstruction vomiting takes place after the stomach has become overfilled and it is projectile in character. The vomiting of intestinal obstruction is forceful and persistent; at first, gastric contents are rejected and later fecal matter appears. The vomiting of brain disease (reflex vomiting) is projectile and unassociated with any gastric derangement. *Cyclic vomiting* is periodic; it occurs in older children and is associated with symptoms of acidosis.

For purposes of *chemical examination* the gastric contents may be recovered by means of a catheter. Residual food will be found in cases of dilatation of the stomach and in pyloric stenosis. Free hydrochloric is normally present in the gastric contents but cannot be demonstrated after a feeding of milk because the milk combines with the hydrochloric acid forming an acid albuminate. The tests for occult blood should be made when the vomitus presents the characteristic appearance of coffee-grounds. In serious nutritional disturbances free hydrochloric acid may be absent.

ACUTE GASTRIC INDIGESTION; DYSPEPSIA.

An attack of indigestion in an infant or in a young child may be accompanied by a train of general symptoms which are at times of an alarming character. As a rule there is only noted the general discomfort, nausea and vomiting caused by the presence of undigested food in the stomach. In the more serious cases, however, fever, drowsiness, fetid breath and headache are also observed due to the toxemia associated with the condition. Acetone and diacetic acid may be found in the urine as well as a trace of albumin. In children of a certain type convulsions are apt to set in, or severe uncontrollable vomiting may occur. Ordinarily, emptying of the stomach and intestinal tract gives speedy relief of the symptoms but in some cases the toxic manifestations persist for some time after this has taken place.

Etiology.—Indigestion is a frequent disturbance in young infants that are artificially fed. The digestive powers of the stomach are feeble and it is not very tolerant to any food excepting woman's milk. If food be taken in larger quantity or in more concentrated form than the stomach can manage, it will not be digested; irregularity in feeding, especially too frequent feeding, is also a prolific cause of indigestion. If the milk is not fresh it may produce serious toxic symptoms.

In older children the same exciting causes which are active in adults are frequently found. Irregularity in eating is a common cause of indigestion in children, and since they are apt to overeat, they frequently suffer from attacks of indigestion. Improper mastication of the food, usually due to bad teeth or malocclusion must always be considered as a possible cause. Lack of proper attention to the bowels and indulgence in the articles of diet which are especially forbidden to children, such as nuts, fruit-cake, cheese, pastry and cheap candy is also frequently noted as an etiological factor. Some children have a subnormal tolerance for sugar and fats and they are consequently readily upset by candy and ice cream even when these

are of good quality. Raw fruits must also be given to children with caution and when fruit is permitted it is most essential that it be clean and neither under- nor over-ripe.

Symptoms.—In infants the first symptoms that will attract attention are restlessness, crying and vomiting. The vomited matter consists of curds, undigested or partially digested food, as the case may be, and it is usually sour and mixed with mucus. The acidity is mainly due to the presence of an excess of lactic acid.

Should the stomach not empty itself completely, severe constitutional symptoms may occur from the absorption of products of incomplete digestion. The infant develops a high fever, becomes apathetic and prostrated; the tongue is coated, the epigastrium bloated, and diarrhea supervenes. This condition may be associated with general convulsion.

Older children are usually feverish, complain of headache, nausea, and gastric pain; the advent of vomiting brings relief. The vomitus may contain food that has been in the stomach many hours.

A mild attack of indigestion may subside spontaneously after the stomach has been emptied either by vomiting or fasting; in the more severe attacks troublesome vomiting and toxemia or an acidosis may be associated. The child's digestion may be impaired for a long time after such an attack. A proper regime of diet and general treatment is necessary in such cases to prevent recurrences or a protracted digestive disturbance.

Treatment.—The management of dyspepsia in the artificially fed infant is discussed in the chapter on "Infant Feeding." In the more serious cases lavage of the stomach should be resorted to.

In older children lavage is not readily employed and we should encourage vomiting by giving copious draughts of lukewarm water to which some bicarbonate of soda has been added. If the symptoms are serious and the child cannot vomit an emetic should be used. The bowels should also be promptly

moved by means of a low enema of soap and water followed by a high irrigation with normal salt solution if fever and toxemia are present.

Remedies are seldom necessary in infants after the stomach has been emptied and the diet carefully regulated. Infants with habitual indigestion, however, will require a remedy such as *ippecac*, *nux vomica* or *pulsatilla*. The same holds good for older children. One of the following remedies will be found useful during the attack. In older children, who can relate their symptoms, we are often capable of averting an attack by an early prescription.

Abies nigra.—Sensation of a hard-boiled egg in the stomach.

Antimon. crud.—Tongue white, heavily coated; great nausea; results of overeating.

Arsenicum.—Indigestion after chilling the stomach. There is severe gastralgia; nausea and vomiting; craving for water which is taken in small sips at frequent intervals; prostration; albuminuria.

Bell.—Throbbing headache; strawberry tongue; convulsions. It is the best remedy for the febrile case with toxemia.

Bry.—During summer and sultry weather; anorexia, thirst for large amounts of water; mouth dry; distress and pain in stomach, as of a load.

Ipecac.—This is the most important remedy for persistent nausea and vomiting.

Nux vomica.—This is the best remedy for the ordinary case of indigestion brought on from overeating. There is a bad taste; the tongue is coated; breath offensive; nausea, with ineffectual efforts to vomit; belching; gastric distress; headache and constipation.

Pulsatilla is indicated in subacute cases of indigestion in children suffering with post-nasal catarrh; coated tongue; delicate digestion easily upset by any rich food. Mild, nervous type of children.

CHRONIC INDIGESTION.

The term chronic indigestion is applied to a group of cases in which a disturbance of the digestive function with resulting malnutrition are the leading clinical features. In some instances gastric symptoms predominate while in others the intestinal tract is chiefly affected. In the majority of instances it is, however, impossible to draw a sharp line of distinction between the symptoms which result from a deranged gastric function and those due to the associated intestinal disturbance. It is well to bear in mind that we are usually dealing with a child whose entire alimentary tract is in a subnormal condition and that under such circumstances there is no practical value in attempting to differentiate between a gastric indigestion and an intestinal indigestion.

The results of chronic indigestion are far reaching upon the child's general health. Primarily, malnutrition and anemia develop from a failure of the proper digestion and assimilation of the food. The child becomes irritable and peevish and may develop serious nervous symptoms. The latter are either due to the malnutrition or they may be toxic in character from an intestinal toxemia.

The dyspeptic symptoms which are commonly observed in these cases are loss of appetite or a perversion of the same; distress after eating and other gastric symptoms such as belching, nausea, vomiting, heartburn and pylorospasm; intestinal flatulency and abdominal pains; diarrhea or constipation.

Etiology.—Chronic indigestion may result from long-continued dietetic errors especially when these date back to infancy. Overfeeding or the use of a food not suited to the child's age, such as fat or starchy foods in excess may result in serious digestive disturbances difficult to eradicate. In older children an improper diet is frequently found responsible for the indigestion or we may find that the child is drinking excessive quantities of milk, especially with its meals, or eating

between meals, and thus overtaxing its digestion. There may be a subnormal tolerance for certain food elements, notably for fat and sugar. Such children are likely to develop attacks of recurring vomiting or fever of intestinal origin when they overstep the tolerance for these foods. There is a growing tendency to look upon some of the chronic digestive disorders, especially those of intestinal origin as forms of deficiency diseases (McCarrison, *Studies in Deficiency Diseases*, 1921).

Constitutional causes are frequently operative. A neuropathic or a gouty heredity; a frail constitution with the "Stiller type" of build, namely a long, narrow chest with acute costal angle, marked mobility of the tenth rib and predisposition to enteroptosis, are strong predisposing causes.

Chronic infections like tuberculosis and focal infections in the tonsils and teeth may be etiological factors. Some cases date back to an acute infection of the gastrointestinal tract from which the child has never fully recovered but which marks the beginning of the present trouble. Carious teeth and malocclusion should always be looked for in chronic indigestion.

Mechanical factors are atony of the stomach; dilatation and ptosis of the stomach; ptosis of the colon; dilatation of the colon; long, angulated sigmoid.

Dilatation of the stomach may result from dietetic errors in infancy or from pyloric obstruction during infancy or it may develop as a symptom of rickets. In older children it can develop from overloading the stomach, especially if there is *gastric atony*. Ptosis also may lead to dilatation by causing a delay in the emptying time of the stomach. An atonic stomach readily dilates if kept continuously overfilled and serious toxic symptoms may occur in conjunction with food retention in such a stomach.

Intestinal stasis from a ptosed or dilated colon or an elongated sigmoid may result in the development of an *intestinal toxemia*. The symptoms referable to intestinal toxemia are varied, prominently among which may be mentioned nervous irritabil-

ity; headache; disordered sleep; enuresia; recurring fever; chronic skin eruptions. Kerley (*Amer. Jour. Diseases of Children*, April, 1920) reports a series of interesting roentgen-ray studies in children with gastrointestinal disorders and points out the importance of making use of roentgenograms in the diagnosis of these affections. The abnormalities which were demonstrated were pylorospasm; dilatation and ptosis of the stomach; dilatation of the colon; ptosis of the colon and elongated and angulated sigmoid.

Symptoms.—The symptoms will depend largely upon the type of case, that is, whether the trouble is chiefly dietetic or whether we are dealing with a condition which is mainly mechanical in nature. Cases in which gastric symptoms predominate will show decided disturbances in the appetite and if there is atony, gastric splashing can be demonstrated in the stomach region. As a rule there is distress in the epigastrium after eating and tenderness to pressure in this region. When there is ptosis with delayed emptying and retention of food recurring attacks of vomiting frequently occur. In the intestinal form there is diarrhea; peristaltic unrest; abdominal pains; mucus in the stools together with undigested fat and vegetable detritus and usually abdominal distention.

Fat indigestion is the predominating feature in some cases. These are characterized by the presence of abnormal amounts of fat and fatty acids in the feces, the stools being loose and acid in reaction. The underlying cause is usually a pancreatic insufficiency.

In carbohydrate indigestion the stools are liquid, of a gruel-like consistency and yellow color, they are frothy, due to fermentation and their reaction is acid. There is little or no mucus. Undigested starch granules can be demonstrated by microscopic examination. There is usually much gas in the intestines and the abdomen is distended. The severe type of intestinal indigestion in which there is both fat and carbohydrate indigestion with associated malnutrition and prominent

abdomen is clinically best known under the name of **coeliac disease**.

Mucous colitis is usually associated with protein indigestion; at least these cases are benefited by a diet from which proteins are excluded and which consists chiefly of green vegetables and cereals. In mucous colitis there is a tendency to constipation; the stools are large and formed and are usually coated with tenacious mucus. At times mucous casts of the intestine are passed with colicky pains. Nervous symptoms and malnutrition are associated.

Infantilism.—There is a type of malnutrition with stunted growth due to chronic intestinal indigestion, which was first described by Herter and named by him Intestinal Infantilism. Children presenting this condition are stunted in growth; the abdomen is large; the stools are abnormally large, containing an excess of fat and fatty acids and the bacterial flora is predominatingly Gram-positive. A marked indicanuria is also observed.

Diagnosis.—The diagnosis of chronic indigestion particularly of the intestinal type, can usually be made from the appearance of the child alone. The malnutrition, anemia, stunted growth in cases of long standing and the prominent abdomen are quite characteristic. The condition of the bowels is also of diagnostic value. Diarrhea is usually present in the earlier stages; the stools being large and containing, beside undigested food particles, an abundance of mucus. They are abnormally offensive and when fermentation is a prominent feature they are acid in reaction and are expelled with a great deal of gas. Undigested fat is present in large amounts as well as some unaltered starch when this has entered largely into the diet. In the later stages the stools are apt to become constipated, usually being pale in color and abnormally large. The stool is often coated with mucus.

In young children the differential diagnosis between *chronic intestinal indigestion* and *tubercular peritonitis* must be made.

The latter is excluded by the absence of a continuous fever, the abdominal tympany without evidence of fluid or enlarged mesenteric glands and the negative von Pirquet reaction.

Hirschsprung's disease presents enlargement of the abdomen due to dilatation of the colon but this is a congenital affection, and a history of obstinate constipation with enlargement of the abdomen dating back to early infancy can be obtained. Furthermore, in Hirschsprung's disease there is hypertrophy as well as dilation and the thickened walls of the colon can be distinctly felt while peristaltic waves are usually clearly visible.

In older children it is important to eliminate other diseases such as chronic appendicitis, tuberculosis, focal infections and intestinal parasites in the diagnosis of a primary digestive derangement. The X-ray is of the greatest value in demonstrating the mechanical conditions which are responsible for many of these cases.

Treatment.—The treatment must be both hygienic and dietetic. Since the digestive function is so largely dependent upon a good supply of blood and draws upon the energy of the organism to a large extent, fresh air and ample sleep are of great importance. These children are usually poor sleepers; they should therefore be made to rest at least half an hour after every meal. Proteins, especially beef-juice, finely minced rare meat and albumin milk (Eiweissmilch) are well born. Butter-milk is also a good food in these cases. Strained green vegetables are useful and together with beef-juice help to overcome the anemia. Since fat and carbohydrate indigestion are the leading features of the majority of cases a high protein diet is, as a rule, useful. In cases with intestinal toxemia, especially constipated cases with stasis or ileocecal regurgitation and in cases of mucous colitis, a carbohydrate and vegetable diet must be employed. All cereals must be cooked for several hours and milk should be boiled. Often preparations like Dryco and

Malted Milk agree better than fresh milk. Zwieback is better digested than bread or toast.

Irrigation of the bowels is of value in the cases with loose, offensive stools and constipation should be treated with massage and low enemata rather than with laxatives. Liquid paraffin is sometimes of distinct value in the constipated cases.

An abdominal support will be required, for a time at least, in most cases.

Cases in which constipation is present will be benefited by one of the following remedies, selected upon its characteristic symptoms:

Calcareo carb. Rachitic and scrofulous children; fair complexion, tendency to perspire. Prominent abdomen; clay colored, putty-like stools.

Carbo veg. Gastric flatulency; sour eructations; slow digestion.

Hydrastis Canadensis. This is a good bitter tonic in small doses and is especially useful in chronic catarrhal affections of the gastrointestinal tract with atonic dyspepsia and constipation with mucus in the stools.

Lycopodium. Lithemic cases; distended abdomen; hard constipated stools; urine shows a brick dust deposit or uric acid crystals. Child has a capricious appetite and is very fretful.

Mercurius vivus. Tongue heavily coated; foul breath; chronic nasopharyngeal catarrh; slight enlargement of the liver; pale, grayish stools.

Nux vomica. Gastric symptoms predominate. Distress after eating; poor appetite; headache and nausea frequently occur. The child is nervous and fretful; abnormal appetite at times preceding attacks of indigestion; recurring vomiting.

When diarrhea is a characteristic feature the following remedies will be called for:

Calcareo phos. Anemia and malnutrition; sunken flabby abdomen; green, slimy, fetid stools.

Mercurius corrosivus. Dysenteric type of symptoms. Blood

frequently present in the stools. The lower colon is chiefly involved.

Magnesia carb. Sour, frothy stools containing large amounts of undigested fat particles.

Phosphoric acid. Painless, watery stools; abdomen greatly distended. There is marked prostration and exhaustion.

Sulphur. Chronic diarrhea, worse in the early morning hours. The stools are watery and irritating. Chronic skin affections are associated.

ACIDOSIS.

The term acidosis indicates a decrease of the alkaline reserve of the blood plasma. There are no characteristic clinical symptoms to indicate such a condition until a dangerous degree of hydrogen-ion concentration of the blood has developed. Under these circumstances the characteristic breathing of air-hunger sets in, the breath may betray the odor of acetone and coma eventually develops. When acidosis has progressed to this stage it is usually beyond medical help.

The earlier stages of acidosis and the compensated form must be diagnosed entirely by laboratory methods. The term "acidosis," being a catchy phrase, has unfortunately become a popular diagnosis and now occupies the place in medicine which such phrases as "uric acid diathesis" and "autointoxication" formerly held. Not that we wish to cast any reflections upon the validity of using the term acidosis in its proper place, however many conditions have been attributed to an acidosis without the slightest proof that such a condition was present. On the other hand, cases with a serious reduction in the blood bicarbonates, either due to renal insufficiency or to the accumulation of acetone bodies in the blood have been overlooked because the proper tests for determining this condition were not made.

The important fact, then, to bear in mind in conjunction with acidosis, is that there are no reliable clinical signs upon which acidosis can be recognized until it has developed to a dangerous

point. We must therefore be thoroughly conversant with certain laboratory methods which are both practical and valuable in the early recognition of an increase of acid radicals in the blood plasma.

One of the simplest tests is that devised by Palmer and Henderson. This test is based upon the fact that when the normal buffer substances in the blood plasma, namely the bicarbonates and phosphates, are more or less exhausted by the accumulation of non-volatile acid substances in the blood, the individual's alkaline tolerance rises. In a normal individual four to five grams of bicarbonate of soda cause the reaction of the urine to become alkaline while in acidosis much larger quantities are required.

Acidosis occurs when non-volatile acids, the result of a perverted metabolism, accumulate in the blood beyond the point at which they can be promptly eliminated or neutralized by the buffer substances in the blood plasma. The commonest acids encountered in acidosis are the acetone bodies. These are derived from the fatty acids of the food fats, or in some cases from the body fats themselves, being intermediary products of incomplete combustion. Normally fats are oxidized into carbon dioxide and water; under certain abnormal conditions of carbohydrate metabolism, or in certain toxic states, the acetone bodies accumulate in the blood. The same thing may happen in carbohydrate starvation for it has been determined that "the fats are burned in the flame of carbohydrate combustion." This explains why acidosis frequently develops in a diabetic when he has been too abruptly deprived of carbohydrates.

In the early stages of such an acidosis, acetone alone appears in the urine, since this substance is nearer to complete oxidation than the other acetone bodies and is therefore the first evidence of a failing combustion of fats. Acetonuria is frequently detected in febrile disturbances and digestive derangements and is not necessarily a serious condition. If the inability to oxidize fats, however, becomes still more pronounced then the more

dangerous acetone bodies, namely aceto-acetic acid and oxybutyric acid make their appearance and a serious increase of acid radicals with a corresponding decrease of carbon dioxid in the blood plasma occurs. When the H-ion concentration of the blood increases beyond a certain point there is a corresponding inability of the blood to take up carbon dioxid. Consequently air hunger and increased respiration sets in, without, however, a cyanosis, as usually exists with the dyspnea of respiratory affections.

With the development of such an acidosis from the production of acids in the process of metabolism, ammonia is formed for the purpose of restoring bicarbonate and alkaline phosphate from the products of the reaction of these substances with acids, or for the purpose of directly neutralizing acids. This ammonia is derived from the products of protein catabolism, these being utilized in the form of ammonia instead of being converted into urea.

Another factor in combating the development of an acidosis is the conversion of alkaline phosphate into acid phosphate. This is excreted by the kidneys in excess of alkaline phosphate and thus tends to restore the alkalinity of the blood.

Finally, an increased excretion of carbon dioxid by the lungs is brought into play. When the blood carbonates are reduced the respiratory center is stimulated, breathing is augmented and an increased elimination of CO_2 results. The decrease in carbonic acid may compensate for the increase of the non-volatile acids and an acidosis is thus averted for the time being. When, however, the alkaline reserve has been entirely depleted overstimulation of the respiratory center occurs with hyperpnea and a marked fall in the carbon dioxid tension of the alveolar air. If the alkaline reserve cannot be replenished at this stage the acidosis reaches a point which is incompatible with life.

From the clinical standpoint we can recognize two degrees of acidosis and also two distinct clinical types. The commonest is the compensated form in which the excess of acid in the

organism is met by an increased excretion of acid sodium phosphate by the kidneys, an increased elimination of carbon dioxid by the lungs and the production of ammonia in the catabolism of nitrogenous foods. Such a condition can be demonstrated by laboratory methods but from the clinical standpoint it is not an acidosis. However, it may be converted into an active acidosis through the occurrence of an acute infection or an intestinal toxemia which break down for the time being the compensatory mechanism. In a child with a normal metabolism such factors would not be able to precipitate an acidosis.

The nature of the metabolic disturbance underlying the development of attacks of acidosis is not understood. There are the indications of sub-oxidation and of intolerance for normal amounts of fat. There is also a prominent nervous factor in many cases and indications of chronic intestinal toxemia. The commonest dietetic error is overeating. Focal infection, especially diseased tonsils and adenoids are commonly found in children who are prone to acidosis. A family predisposition is often noted. The acidosis which develops in infants as a complication of a severe diarrhea is due to the rapid loss of water and alkali through the intestines and the associated toxemia and renal insufficiency.

There are two clinical types of acidosis; the one is apparently a *primary* condition while the other occurs as a *secondary* manifestation of severe forms of *infantile diarrhea* or of *cyclic vomiting*. In both of the latter forms there is presumably a previous relative acidosis which is converted into an active, or manifest acidosis by the above mentioned disturbances.

In speaking of "acidosis," or "acid intoxication" the first class of cases is usually implied. While the acidosis is the predominating clinical condition, still these patients almost invariably show evidence of an infection, usually of the upper respiratory type, preceding the symptoms of acidosis. No doubt the toxemia due to the infection is responsible for the

increase of acetone bodies in the blood. In this respect the acidosis differs from that observed in cyclic vomiting and diarrhea in which cases it is of later occurrence and is due to the acute inanition, loss of body fluids and interference with the renal function.

The characteristic symptoms are fever ranging from 102° to 104° F.; a mild type of tonsilitis or nasopharyngitis; vomiting and hyperpnea. The degree of acidosis is usually mild but serious and fatal cases occur. There is a strong tendency to recurrence as in cyclic vomiting and frequently several children in the same family are affected. For treatment see "Cyclic Vomiting."

CYCLIC, OR RECURRENT VOMITING.

Cyclic vomiting is a condition in which periodic attacks of uncontrollable vomiting occur. It is most frequently encountered in children between the ages of three and six years, the sexes being affected about equally. Persistent vomiting developing in an infant should suggest the onset of tuberculous meningitis rather than this condition, although vomiting and acidosis may develop secondarily to a severe infantile diarrhea. Acidosis has in fact been looked upon as the cause of cyclic vomiting, owing to the fact that acetone and diacetic acid can usually be detected in the urine during an attack. The presence of the acetone bodies in the urine must, however, be looked upon as a secondary condition, because they are often absent at the time of the onset of the vomiting, sometimes not showing before the second or third day, and also because they are never present in large amount.

The primary cause is no doubt a metabolic disturbance with diminished oxidation. This leads to the accumulation of unoxidized toxic substances in the blood. The nature of these substances is not known. In the severe types of cyclic vomiting albuminuria, casts, and a leucoecystosis occur in conjunction with the vomiting. A derangement of the glycogenic function of the liver may be responsible for a disturbance in the oxidation

of the fats so that acetone, diacetic acid and oxybutyric acid appear in the blood as intermediary bodies of the incomplete catabolism of fats. It is evident that any factor which may upset the glycogenic and perhaps also the detoxicating function of the liver, can become an important indirect cause of an acidosis. Overeating, intestinal toxemia, nervous excitement or shock, over-fatigue, and bacterial toxemia are all to be considered in this light and their etiological relationship to cyclic vomiting is to be explained on these grounds. It is a common clinical observation that many children who are subject to these attacks have chronically infected tonsils and adenoids. An acute infection in such a child will result in a more pronounced reaction than in a normal one and may thus precipitate an attack. Nervous excitement and fatigue also frequently precipitate an attack perhaps through the derangement of the delicate mechanism of the hepatic function which is under nervous control. Indeed, some clinicians have looked upon cyclic vomiting as a manifestation of hysteria because the nervous element plays such a prominent role in its etiology. Whether such a condition as chronic appendicitis acts as a reflex cause or in the nature of a focal infection is difficult to decide. Personally I have seldom found symptoms referable to the appendix. Kerley believes that many cases of recurrent vomiting are due to a dilated or ptosed stomach in which there is retention and residue long after the feeding period as demonstrated by Roentgen-ray studies and that acidosis has nothing to do with their etiology. Personally I have encountered cases in which acidosis could be positively demonstrated while in others there was an absence of acetone bodies in the urine throughout the attack together with normal carbon dioxid tension of the alveolar air. It is indeed surprising that the more serious symptoms of acidosis are so seldom encountered in cyclic vomiting. No doubt the early onset of vomiting and the absolute fast which the patient is forced to undergo tends to bring about a spontaneous cure of the case. The fact that the attack is self-

limiting, the vomiting being uninfluenced by any mode of treatment, and that prompt recovery is the rule, tend to substantiate this belief.

Symptoms.—Prodromal symptoms, such as anorexia, malaise and slight fever may be present, but they are frequently absent and the attack occurs abruptly after some form of nervous excitement or a "cold."

Vomiting is the chief symptom, being persistent and apparently not traceable to acute indigestion. It usually continues for from two to three days, being little influenced by treatment. There is no pain and the abdomen is soft and retracted. Nausea is slight or absent.

The bowels are constipated and the colon may be felt in a state of spastic contraction. The stools are sometimes light in color. As a rule there is no fever excepting in the early stages.

The child's general condition may become alarming although convalescence is usually rapid and fatalities are rare. Prostration is marked and the child lies in an apathetic state suffering intense thirst but unable to retain even a swallow of water. The tongue becomes dry, the lips are abnormally red, the eyes sunken, the respirations are shallow and the pulse is exceedingly rapid. Air-hunger does not occur in the milder cases but may become quite marked in the severe forms of the disease. The pupils and reflexes are normal and Kernig's sign or other evidence of meningeal irritation are never noted. A characteristic fruity odor can usually be detected on the breath, indicating the presence of acetone. At this stage the urine becomes scanty and contains varying amounts of acetone and diacetic acid.

The milder cases subside at the end of forty-eight hours. They are generally encountered in neurotic children, coming on, as a rule, after overexertion, fright or excitement. Indiscretions in diet and neglect of the bowels may, of course, be preceding factors.

Occasionally a more serious type is encountered in which the

vomiting may persist for from four to five days with the development of symptoms of acidosis and grave prostration. A fatal outcome occurs at times. The urine in these cases shows albumin, blood and casts beside the acetone bodies and the blood shows a leucocytosis. Hematemesis is usually present, adding to the gravity of the case.

Diagnosis.—In mild cases a first attack may not be recognized as a distinctive type of vomiting but a recurrence in the course of several months without the history of a dietetic error should arouse suspicion of the true nature of the attack.

Simple *nervous vomiting* frequently occurs in nervous children after excitement or overexertion, but it is of short duration and there is only the vomiting of food.

Acute gastric indigestion gives a history of an indiscretion in diet and the symptoms promptly improve after the stomach and bowel have been emptied while in recurring vomiting the retching and vomiting persist in spite of all treatment directed to the stomach; in other words, it acts precisely as other forms of toxic vomiting, like the vomiting of pregnancy, for example.

Appendicitis and *intussusception* can be excluded by making a careful abdominal examination, supplanted, if necessary, by a rectal examination. In intussusception we encounter a sausage-shaped tumor in the abdomen together with hyperactive peristalsis and often a bloody discharge from the rectum. In appendicitis there is fever and abdominal pain together with tenderness and rigidity over the appendix or a sensitive mass in the appendicular region. If septic peritonitis develops the abdomen becomes distended, peristalsis ceases and the pulse rate is disproportionately high. At this stage the vomiting may become stercoraceous, which it never does in recurrent vomiting.

Tubercular meningitis, in the early stages, may closely simulate the picture of recurring vomiting. There is the mental apathy; the vomiting without apparent cause and retracted, non-sensitive abdomen. The pulse and respirations are, however, more irregular in meningitis; involvement of the ocular

muscles soon makes its appearance and Kernig's and Babinski's signs are found even in the early stages of most meningeal conditions. Finkelstein cautions against excluding brain affections because of a history of previous attacks of vomiting, as such conditions may present repeated attacks of intracranial pressure with clear intervals. An examination of the eye-grounds is of great value in such cases.

Acute nephritis cannot be ruled out at the height of an attack when albumin and casts are found plentifully in the urine, but the absence of edema and of suppression of urine and the rapid disappearance of the albumin and casts after the subsidence of the attack exclude a primary nephritis.

Treatment.—Treatment between attacks should first be considered. As these children are usually of a neurotic temperament and present evidence of defective elimination they must be kept on a diet in which milk, cereals, whole-wheat or bran bread, fresh vegetables, and fruit play the major role. Fat should be prohibited, and sugar and starch given sparingly, although not too rigidly excluded because of the importance of carbohydrates as heat-producing foods and their role in the metabolism of fats. Meat may be taken sparingly. The chief thing to guard against is overeating. Intestinal autointoxication is to be strictly prevented and whenever indican appears in the urine in excess the child should receive a saline laxative and an effort made to have it drink buttermilk instead of sweet milk. Systematic colon irrigation should be practiced in cases with iliac stasis.

The administration of small doses of bicarbonate of soda—five grains three times daily, after meals—between attacks has proven of some value in lessening the frequency of the attack when the above dietetic precautions are observed.

Remedies, such as *calcarea carb.*, *calc. phos.*, *lycopodium*, *ignatia*, *arsenicum*, etc. are useful in correcting chronic disturbances in the digestive tract or nervous manifestations of long standing. A remedy which I have found useful in migraine in lessening the number of attacks and which is also applicable

to cyclic vomiting is *sanguinaria* which should be administered in doses of several drops of the tincture or 1x dilution after meals.

During the attack the child should be immediately put to bed in a quiet, partially darkened room and all food withheld for the first twenty-four hours. Cracked ice may be permitted and the child should be urged to take half a glass of water containing twenty grains of bicarbonate of soda. If this is vomited it should again be tried in an hour when it may possibly be retained. Should the second attempt fail, a few teaspoonfuls of the soda solution may be administered every half hour and if it is retained the amount may be increased. When, however, all attempts to give water or bicarbonate of soda by mouth result in vomiting, the best course to pursue is to discontinue everything by mouth and give normal salt solution by rectum. If the vomiting persists after the second day, three per cent of glucose may be added to the normal salt solution and given by enteroclysis.

On the second or third day such foods as albumin water, peptonized milk, barley-water and strained rice broth may be attempted, given in small quantities at two hourly intervals. At times, a gruel, such as farina or cream of wheat, or solids like zwieback are better retained than liquids.

Remedies.—*Nux vomica* and *ipecac* may be useful early in the attack. The indications for *nux vomica* are headache, nausea, constipation, coated tongue, delayed digestion (stasis) or ill effects of overeating.

The indications for *ipecac* are chiefly the persistent vomiting without signs of a gastric disturbance.

Small doses of *calomel*, $\frac{1}{10}$ to $\frac{1}{20}$ grain, every hour for ten doses at times relieve the vomiting and at the same time act as a mild purgative.

Belladonna is useful when there is fever and when the attack is of nervous origin.

Arsenicum is indicated in the severe forms of vomiting associated with albuminuria and marked prostration.

Bromides, given by rectum, are also useful in the more severe type of cases.

HYPERTROPHIC PYLORIC STENOSIS.

Pyloric stenosis occurs in infancy as a result of a congenital hypertrophy of the musculature of the pylorus. The stenosis is progressive in development and may terminate in complete obstruction. The circular muscular fibres are mainly involved; other pathological changes are wanting. Muscular spasm, no doubt, plays a part in the production of the pyloric obstruction and cases of pylorospasm unaccompanied by a hyperplastic tumor involving the pyloric muscle are by no means rare and must be considered in the diagnosis of pyloric stenosis.

Pyloric stenosis is more frequently encountered in male than in female infants. It has no connection whatsoever with dyspepsia or improper feeding; indeed the majority of cases appear to develop in breast-fed infants. The *etiology* is unknown; a family predisposition is noted in some cases. Two of my cases occurred in the same family. The condition is a congenital malformation probably atavistic in type.

Symptoms.—The babe is usually well developed and well nourished at birth, nursing and gaining normally for a time. Vomiting is the chief symptom and the first one to call attention to the condition. It comes on more or less abruptly toward the end of the first month and gradually increases in severity and in persistency so that the child loses weight rapidly and the bowels become obstinately constipated. Enemata bring away bile-stained mucus but no milk residue. The food is naturally suspected of disagreeing with the infant but dietetic treatment exerts no influence upon the vomiting which now occurs practically after every feeding and becomes projectile in type. Sometimes one feeding is retained but after the next feeding the combined amount of both is vomited. Vomiting is not accompanied by nausea or pain. The babe may vomit toward the end of a nursing and then eagerly seeks the nipple and nurses all over again.

Dilatation of the stomach gradually takes place together with hypertrophy. The epigastric region is prominent, especially after nursing, while the lower abdomen is sunken and flaccid. The intestinal tract is practically empty. When the stomach is filled, peristaltic waves make their appearance due to the abnormal effort of the stomach to empty itself. These waves originate in the left hypochondriac region and cross the epigastrium to the region of the pylorus where they cease. The outline of the stomach is plainly indicated by these waves and we can observe from day to day the increasing gastric area.

At this stage a small oval tumor about the size of a small olive can be demonstrated in the pyloric region in the majority of cases. The usual site is to the right of the median line, midway between the tip of the xiphoid and the umbilicus. It is most readily felt just after vomiting, at which time the abdomen is relaxed.

Residual food is usually present in the stomach and this is a most important diagnostic sign. By inserting a No. 16 French soft rubber catheter into the stomach four hours after a feeding, an ounce or two of residual food can be aspirated, providing there has been no previous vomiting. The X-ray also shows the opaque meal still in the stomach and the cap and duodenum empty.

The *prognosis* and ultimate outcome of cases of pyloric stenosis depend upon the degree of anatomical changes present in the pyloric region of the stomach and whether the symptoms are predominatingly mechanical or spasmodic in character. Owing to the difficulty of sharply differentiating hypertrophic stenosis from pylorospasm, confusion as to the prognosis naturally arises. Undoubtedly many cases of spasmodic pyloric stenosis have been mistaken for hypertrophic stenosis. Again, a case of hypertrophic stenosis may at times present more spasm than actual anatomic obstruction and so recover under medical treatment. Cases, however, which show an absolutely unyielding obstruction at the pylorus and in which a tumor can be distinctly felt, do not yield to any but surgical treatment.

The *diagnosis* of pyloric stenosis rests upon the history, the persistent projectile vomiting, the gastric dilatation with visible epigastric waves and the progressive rapid emaciation of the infant. The first symptom is vomiting, setting in abruptly in an infant from two to four weeks old without assignable cause. Associated with the vomiting is obstinate constipation and scanty urine and loss of weight. There is no evidence of nausea or pain and dietetic treatment does not influence the vomiting.

Pylorospasm is differentiated by the irregularity in the character of the vomiting, the fact that food still passes into the intestines as shown both by the character of the stools and by the X-ray plates. A tumor can at times be felt in spasmodic cases due to spastic contraction of the pylorus, but this disappears after vomiting or after gastric lavage. Furthermore, medical and dietetic treatment bring results which are not seen in hypertrophic stenosis.

Treatment.—The early treatment of the vomiting is that of dyspeptic vomiting. Milk should be discontinued for a time, and whey, barley-water, and albumin-water may be tried. Lavage should be systematically carried out. The stomach should be thoroughly washed out once or twice daily with a warm solution of bicarbonate of soda, one drachm to the pint, for the purpose of removing all food residue and to wash out the mucus. The child should be nursed at four hour intervals and heat applied to the epigastrium after each feeding. The child should be placed on its right side with the head slightly elevated. If, in spite of these measures, breast milk is not retained we should try the thick farina treatment advocated by Sauer of Chicago, (*Archives of Pediatrics*, July, 1918).

The formula recommended by Sauer is made up as follows:

Skimmed milk	9 oz.
Water	12 oz.
Farina	6 tablespoonfuls.
Dextri-Maltose	3 tablespoonfuls.
Boiled for one hour in a double boiler.	

At each feeding the required amount should be warmed and then administered on the end of a spoon handle. The infant may be slow in swallowing the thick paste and it sometimes takes an hour to give the required amount. Two to three ounces may be fed every four hours. As a rule, the infant soon learns to take the food and a little patience is all that is required to carry out the treatment. The results are most gratifying in many instances. It is the only form of medical treatment which offers any hope of averting an operation in true cases of stenosis and should be tried on all cases that are seen early.

Sauer has demonstrated by X-ray studies that the patency of the pylorus is much greater after cereal feedings than it is after a milk-mixture feeding. There is therefore a prompt return of food residue in the intestinal contents in all cases which respond to this treatment. Should, however, the vomiting and constipation persist and the infant continue to lose weight, surgical interference becomes imperative.

The operation of choice is Rammstedt's pyloroplasty. The technique is simple but the operation is a delicate one and requires skill and experience. Gastro-enterostomy has been abandoned by most surgeons. The technique presents many difficulties, the surgical shock is great and the mortality high. My experience has been very discouraging with this operation. With the Rammstedt operation, however, the surgical risk is small. The operation is of short duration and not accompanied by shock. The results are brilliant; vomiting usually ceases at once and so far there has been no return of the stenosis in any of my cases.

DIARRHEA.

There are two clinical types of diarrhea encountered in infancy, one of which is due to some error in feeding or to the activity of saprophytic bacteria in the intestinal tract, while the other results from the action of bacteria which are either primarily pathogenic or which are capable of producing toxic substances in the intestines. The first group comprises

the class of cases which are usually described under the headings of *dyspeptic diarrhea*, *fermental diarrhea* and *acute intestinal indigestion*, while the second group embraces the various clinical types of the so-called "*Infectious Diarrheas*."

DYSPEPTIC DIARRHEA; ACUTE INTESTINAL INDIGESTION.

Dyspeptic diarrhea is the commonest and least serious of the acute intestinal disorders of infancy. It is a frequent manifestation of overfeeding. Hot weather and teething are predisposing factors, but an infant may develop diarrhea at any time of the year if it is fed too often or too much at a time or given a formula with an excess of sugar. Cane sugar and sugar of milk cause diarrhea more readily than one of the maltose preparations, while raw milk is more likely to induce it than boiled milk.

In cases of diarrhea due to fat indigestion the infant usually regurgitates excessively and the stools contain small, soft fat curds.

Sugar dyspepsia produces acid, excoriating stools, often green in color or soon changing to green on exposure to air. In protein indigestion the stools are yellow or brown, offensive, and contain tough casein curds. Curds do not occur when boiled milk is used. Milk that has not been properly kept but which is just on the point of turning sour may produce a severe dyspeptic diarrhea.

Symptoms.—The cardinal symptoms are colicky pains, intestinal fermentation and loose stools. Moderate fever is usually present. The number of stools is seldom more than five to six daily. When the condition is one mainly of sugar fermentation they are green in color, acid in reaction and excoriating to the buttox. They are usually liquid and expelled with considerable gas. When the condition is due to proteolytic bacteria the stools are foul, alkaline in reaction and of a brownish color. When raw milk has been fed the stools frequently contain tough casein curds.

The duration is short; fever and constitutional symptoms are usually slight or absent. A previously healthy child promptly responds to proper dietetic treatment but one whose vitality has been depressed by hot weather may develop serious toxic symptoms as the result of a dyspeptic attack. Again, repeated attacks of dyspepsia eventually lead to impairment of nutrition and may thus predispose to the development of one of the nutritional diseases, such as rickets and scurvy. For this reason the importance of dyspepsia must not be underestimated.

If the diarrhea continues for several days the buttox may become excoriated from the lactic and other lower fatty acids in the stool. Mucus may also appear in the stool as a result of irritation of the intestinal mucosa. The urine becomes ammoniacal and irritating to the skin.

Diagnosis.—The short duration, slight fever or absence of fever, and the character of the stools differentiating simple diarrhea from infectious diarrhea. In hot weather infants frequently have watery, yellowish stools due to the enervating effect of heat and humidity upon the nerves controlling the secretions and movements of the intestines.

The diarrhea ushering in one of the acute infectious fevers can only be identified by the ultimate appearance of the symptoms peculiar to the affection in progress.

Treatment.—Treatment is mainly prophylactic. Starchy foods should not be fed to young infants excepting in weak solution, such as barley water, or dextrinized gruels. Infants whose digestive powers are naturally weak should be fed on proportionately weaker milk formula than robust infants of the same age. During hot weather the fat and sugar should be reduced and it may become necessary to boil the milk in order to prevent the formation of casein curds.

During an attack it is advisable to withhold the usual food for from twelve to twenty-four hours, as necessary, and administer simply boiled water, weak tea, or barley water. On the second day one part skimmed-milk to two parts of barley water,

mixed and boiled for three minutes, may be tried. If this agrees, whole milk should be substituted for skimmed milk. Sugar should be withheld from the food until all signs of indigestion have disappeared. (See Treatment of Infectious Diarrhea, p. 143).

In the diarrheas accompanying teething, or those of a neurotic type, such remedies as *Belladonna*, *Chamomilla*, the *Calcareae* and *Pulsatilla* are the ones usually indicated. For simple dyspeptic diarrhea *Nux vomica* 3x is the best remedy. *Mercurius* 6x is preferable when there is much mucus in the stools and if the buttox becomes excoriated. *China* 2x is useful in atonic diarrhea of hot weather.

Aloes.—Flatulence and rumbling in the lower bowels; large quantities of gas escape with stool.

Bell.—During hot weather and dentition; face flushed, abdomen distended, colicky pains; cerebral symptoms; skin hot and dry.

Bry.—Diarrhea from sudden changes in the weather, especially when there are hot days and cold nights. Diarrhea worse mornings, painful, aggravation from motion.

Calc. carb.—Diarrhea during dentition; rachitic infants; profuse sweating about head; distended abdomen; vomiting and diarrhea. Stools sour and undigested.

Calc. phos.—Dentition delayed; recurring attacks; stools green, with mucus; abdomen flaccid.

Cham.—Dentition; painful, excoriating diarrhea, looking like spinach and eggs. Child cross and irritable; wants to be carried.

China.—Undigested stools; flatulent colic, or painless stool with much fermentation. Anaemia and prostration; loose, yellowish stools in hot weather.

Colocynth.—Pain relieved by firm pressure.

Ipecac.—Green, loose stools associated with vomiting.

Mag. carb.—Stools green and frothy, like frog-pond scum, containing tallow-like lumps. Sour odor; colic relieved by stool; fat diarrhea.

Mercurius.—"It is better indicated the more widely in the departure from the natural color of the motions, and the more slimy they are."—(Hughes.)

Nux vomica.—Acute intestinal indigestion. In the beginning before inflammatory reaction has been set up and when the stools are still strictly dyspeptic in character. Associated gastric dyspepsia.

Podophyllum.—More severe forms of diarrhea. The stools are yellow and watery and are expelled with much gas. Pro-lapsus ani.

INFECTIOUS DIARRHEA

(ACUTE CATARRHAL ENTERITIS; CHOLERA INFANTUM;
ILEOCOLITIS.)

The term "Infectious Diarrhea" is applied to those acute intestinal disturbances which can be traced directly to the action of bacteria or their toxins upon the mucous membrane of the intestines with resulting inflammatory reaction, diarrhea, fever, and toxemia. In this respect it differs specifically from simple diarrhea, or dyspepsia, a purely functional or chemical disturbance, without inflammatory reaction in the gut and toxic manifestations.

An inflammatory diarrhea is, however, not always infectious in the restricted sense of that term. With the exception of the cases of acute ileocolitis presenting characteristic dysenteric symptoms and in which the bacillus dysenteriae can be found in the stools, we have no definite knowledge of the bacteriology of enteritis in infancy. It is true, an enteritis may result from infection of the alimentary tract with bacteria which are capable of setting up local inflammation in other mucous membranes, namely, the nose and throat or bronchi and it is not at all improbable that streptococci, the pneumococcus and the influenza bacillus occasionally induce an enteritis. Such infections, however, are most common in the winter, a time when enteritis is rare. Streptococci are frequently found in the walls of the intestines in fatal cases, but are most likely secondary invaders.

It is also probable that an excessive number of bacteria, not

necessarily pathogenic, entering with the food or developing in the intestinal tract as a result of lowered vitality and dyspeptic conditions, may set up an inflammatory reaction. This can result either from the action of bacterial toxins or from the irritating and toxic effects of products of bacterial decomposition of the intestinal contents. Absorption of these toxic products explains the associated fever and toxemia. Whether such bacteria as the gas bacillus of Welch and the bacillus pyocyaneus play an active role in the cases in which they can be demonstrated in the stools, or are purely accidental has not been finally decided.

Infantile diarrhea is so distinctly a disease of hot weather that the effect of prolonged heat and humidity upon the infant's metabolism and digestion is now almost universally accepted by pediatricists as the primary cause of the intestinal disturbances embraced by the term "infectious diarrhea."

Finkelstein advanced the theory that the diarrhea and accompanying toxic symptoms result from the injurious effect of the sugar in the food, in a child whose intestinal mucosa was previously injured by attacks of dyspepsia and whose vitality had been depressed by hot weather. The sugar is absorbed directly into the circulation, producing pyrexia and intoxication, and can be demonstrated in the urine. A high fat content of the food favors the development of this "Alimentary Intoxication." The salts of the whey have also been shown to produce pyrexia and diarrhea. Czerny and Keller attributed the toxic manifestations to the bacterial decomposition of the food either before ingestion or later in the intestinal tract. They believed the toxic substances to be derived from the fat in the food, this being split into irritating lower fatty acids. The local action of these noxious substances results in an enteritis, while their absorption into the circulation, in conjunction with the depleting effect of the diarrhea, may induce an *acidosis*. Some of the symptoms which were formerly looked upon as toxic manifestations are now recognized as being due to acidosis.

The work of Vaughan with protein split products and his discovery of a protein poison common to all proteins whether bacterial or food-proteins, has shed much light upon the pathogenesis of fever and other symptoms of infection. Some of these observations seem to fit in well with the clinical manifestations of infectious diarrhea. For example, as a result of incomplete digestion, a protein may be absorbed into the circulation in the form of peptone, the stage of digestion at which a protein becomes poisonous (Vaughan). Again, undigested protein may be absorbed from the alimentary tract, in dyspeptic conditions. This may be facilitated by the effect of excessive heat upon the infant's vitality and digestion and in this way some cases of *cholera infantum* might be explained. The fact that sugar is found in the urine, as pointed out by Finkelstein, does not weigh against this theory because albumin is also frequently present in the urine and the blood shows a pronounced leucocytosis.

Bacteria, whether pathogenic or non-pathogenic, can split the proteins of the milk and probably render them absorbable without complete digestion, with resulting acute gastrointestinal symptoms of sudden onset after taking "spoiled milk." These cases are commonly designated *acute milk infection*.

Food.—The importance of food as an etiological factor is only secondary to that of temperature and humidity. Children that are exclusively breast-fed rarely develop enteritis and then probably only through lack of hygienic care. The vast majority of cases occur in infants that are artificially fed and whose food, both as to the quality of the milk and the proper modification of the same, is decidedly below standard. It is always safer to use pasteurized or boiled milk in the summer. Cold weather seems to give a surprising immunity to diarrheal affections even when the quality of the milk is none too good. The investigation of the Rockefeller Institute into the etiology of infantile diarrhea disclosed the rather startling fact that despite the large number of bacteria that were found in many samples

of milk fed to infants in the winter they exhibited a remarkable tolerance for the same. The practice of boiling milk and using pasteurized milk, now becoming more general among all classes, has done much toward reducing the infantile death-rate during the summer months.

The *environment* is an important factor. Fresh air and personal cleanliness are two of the strongest prophylactic measures in infantile diarrhea and when infants are kept in squalid, poorly ventilated or crowded quarters and not regularly bathed they offer poor resistance against an intestinal infection.

Intestinal indigestion predisposes to the development of an infectious diarrhea. All dyspeptic conditions should, therefore, receive prompt attention.

Pathology.—Both local and general pathological changes can be demonstrated in fatal cases. The body is emaciated in appearance due to the great loss of weight from demineralization of the tissues with consequent loss of water. Metabolic disturbances frequently demonstrable during life are sugar, acetone, and diacetic acid in the urine and increased urinary ammonia. Albuminuria, due to acute parenchymatous degeneration of the kidneys is common. The blood shows a leucocytosis.

The liver is enlarged due to fatty changes. Parenchymatous degeneration may be present. The lungs show hypostatic congestion and a secondary bronchopneumonia is common.

In rapidly fatal cases of cholera infantum the gut may show no gross lesions but microscopic examination will reveal degenerative changes in the epithelial cells of the superficial layers of the intestinal mucosa. Typical cases of catarrhal gastro-enteritis present acute inflammatory reaction in the gastrointestinal mucosa. The mucous membrane is congested and covered with mucus. The lymph follicles are swollen but not as markedly as in cases of ileocolitis due to dysentery bacillus infection. In protracted cases irregular superficial ulcers form on the intestinal mucosa.

Symptoms.—The symptoms of infectious diarrhea naturally vary with the physical condition of the infant at the time it is attacked and the severity of the pathological process active in the intestine. Cases of dysentery bacillus infection (ileocolitis) present a characteristic train of symptoms and specific pathological changes in the gut and are therefore described separately.

A typical case of enteritis usually begins with vomiting, fever, colicky pains and dyspeptic stools. The abdomen is distended with gas and the stools are at first acid and contain curds until the bowels have been thoroughly emptied. If no change is made in the diet at this time the acrid character of the stools may persist and the buttox become excoriated. When milk, however, is promptly withdrawn and carbohydrates are also withheld, then the fermentative changes in the gut are replaced by putrefaction and the stool becomes offensive and alkaline in reaction.

Associated symptoms are fever, prostration, gastric irritability and rapid loss of weight. A leucocytosis is usually present and albumin may be found in the urine. Restlessness, nervous irritability, or drowsiness and collapsic symptoms may develop. In the more severe form, serious symptoms of intoxication develop and sugar may be found in the urine.

As the condition advances the stools become more frequent and watery, the odor is putrid and mucus is present in appreciable amounts. The color is either brownish or green; when putrefaction predominates they are brown, foul, watery and alkaline in reaction. Gas is passed freely with the stools. This is usually formed from the carbohydrates which are being rapidly fermented by the abnormal bacterial activity in the gut. Kendall has shown that the colon bacillus is a facultative organism capable of inducing either putrefactive or fermentative changes depending upon the nature of the medium in which it is growing. In symbiosis with the bacillus subtilis it is capable of generating large amounts of gas even in a protein medium.

The duration depends to a large extent upon prompt rational treatment and also upon the child's physical condition at the time of onset of the attack. It will also be influenced largely by the severity of the initial symptoms. Many cases can undoubtedly be aborted. The prompt disappearance of alarming general symptoms together with the vomiting and diarrhea which frequently follows upon the early institution of a starvation diet and purgation or bowel irrigation, has convinced many pediatricists that these cases are purely toxic and not infectious in nature. While this is undoubtedly true in many instances, still there is a large percentage of cases which run their course, similar to any other infection, in spite of the above mentioned treatment.

Cases that have gotten well under way before the offending food has been discontinued, or whose intestinal mucosa has been damaged by previous dyspeptic conditions, may continue to have watery stools with mucus and run a temperature for an indefinite period. In these instances bacteria have very likely penetrated the intestinal mucosa and have also been carried into the general circulation. It may, therefore, be stated that in the average case the fever will last from three to four days, provided the child receives prompt treatment and its physical condition before the attack was favorable. The diarrhea may, however, last longer and a tendency to relapse often exists for some time, necessitating great caution in the feeding of these cases. If the infant's condition was poor previous to the attack the reparative process will require a longer time and fever and diarrhea may persist for a week or two. A persisting fever is a grave prognostic omen as it usually signifies a secondary infection or a complication such as bronchopneumonia.

Cholera infantum is a hyperacute type of gastro-enteritis, usually occurring in infants with previous intestinal disturbances and is rarely seen excepting during extreme hot spells. In some cases it may be due to an *acute milk infection*, or it may be a form of heat stroke.

The child is attacked suddenly with severe, repeated vomiting, high fever and diarrhea. The stools are profuse and watery in character, at first foul, later becoming like water and almost odorless. Rapid loss of weight and collapse follow.

The child becomes semi-conscious, the eyes are sunken and there may be an apathetic staring expression. Sometimes the child becomes very irritable, the neck may be retracted and convulsions set in (hydrocephaloid state).

The symptoms described by Finkelstein as *acute alimentary intoxication* are due to the development of an acidosis as pointed out by Howland. The child becomes drowsy and is only semi-conscious. When aroused it looks about with a vacant stare. The respirations are deep and rapid, due to air hunger, and may suggest pneumonia. The urine contains acetone and diacetic acid and may show traces of sugar. The prognosis in cholera infantum and in acidosis is always grave.

Diagnosis.—At the time of the onset of a case of infectious diarrhea it is not always possible to determine whether we are dealing with a primary gastrointestinal condition or whether the diarrhea and vomiting are symptomatic of some other acute infectious disease. During hot weather, however, this doubt rarely arises. A fever and diarrhea that does not promptly respond to appropriate treatment is not likely to be a primary gastrointestinal affection.

The severe nervous disturbances encountered in cholera infantum and in acidosis may suggest meningitis. The initial diarrhea, however, should rule out meningitis. When a case of enteritis presents a continued fever some complication such as otitis, bronchopneumonia and pyelitis should be looked for.

ACUTE ILEOCOLITIS; DYSENTERY.

Acute ileocolitis is an acute intestinal inflammation due to infection with the dysentery bacillus in which characteristic lesions are produced in the lower portion of the ileum and in the colon. The clinical symptoms are continued fever with toxemia and diarrhea.

The dysentery group of bacilli are the only organisms which have been found definitely associated with a specific intestinal infection in infancy. The "acid type," or the Flexner-Harris type is the organism most frequently found in this country. In some cases of ileocolitis streptococci are found in the stools in large numbers and it is possible that these organisms may set up an ileocolitis, on account of their special predilection for lymphoid tissue. The streptococcus is, however, generally looked upon as a secondary invader.

In mild cases the mucous membrane of the lower ileum and more or less of the entire colon appears congested and swollen. In the small intestine the congestion usually appears in streaks on the folds of the mucosa which are seen to run transverse to the long axis of the gut as it is laid open for inspection. The small intestine is distended with gas and filled with undigested food and greenish mucus, which adheres to the surface. In cases of short duration the colon does not show as pronounced changes as the ileum, but in protracted cases it is always more affected.

In more severe cases the deeper structures are involved, as a result of which there is slight thickening of the intestinal wall from round cell infiltration of the sub-mucosa. The lymphoid structures are swollen and there is congestion about Peyer's patches; the latter may stand out prominently, but they seldom ulcerate as in typhoid fever. The lymph follicles of the colon show the greatest involvement and they stand out on the mucous membrane as small beads—follicular enteritis. When the process has been a protracted one the follicles ulcerate. In severe catarrhal ileocolitis ulceration is more likely to take place irrespective of the lymph follicles and results in the production of variously sized, irregular, superficially situated areas (catarrhal ulceration). Hemorrhage does not result from such ulcers but they offer a port of entry for the development of a general bacterial infection and they always tend to protract the case if they do not hasten the death of the child.

Microscopic examination shows destruction of the superficial

epithelial layer and round-cell infiltration of the mucosa. The blood vessels are engorged and the lymphoid structures swollen. In milk cases the process stops here. In severe cases the infiltration reaches to the muscular layers and necrotic changes take place in the inflamed follicles. The epithelium is densely infiltrated with leucocytes and fibrinous exudate can be demonstrated. This is but a step to the membranous variety.

Membranous colitis presents the most pronounced anatomic changes. It corresponds closely to dysentery as seen in adults, but the membrane is not so thick and ulceration does not occur so extensively. The membrane is practically limited to the colon, its ascending portion and the sigmoid flexure being favorite sites. The membrane is of a dirty-gray color and closely adherent to the mucous membrane contrasting markedly with the deep red congestion of the latter where there is no membrane. The main changes are found in the intestinal wall, which is considerably thickened and rigid. Membrane may extend down as far as the rectum, where it can be seen during life as the child strains at stool.

Follicular ulceration is not uncommon in cases that have run a protracted course. It is especially prevalent in institutional cases and among poorly nourished infants that have suffered from repeated attacks of enteritis.

The ulcers are round, varying in size from a pin point to that of a split pea and represent destruction and excavation of the inflamed solitary follicles. They may be found in both the ileum and colon, but most frequently they are confined to the colon.

In association with the distinctive lesions of ileocolitis it is not uncommon to find bronchopneumonia as a complication, which, in fact, may prove to be the determining cause of death. It is usually of the desquamative type; rarely septic, although a general infection from the intestines is possible.

In the kidneys we may find evidence of acute parenchymatous degeneration. Acute nephritis is rare. Similar changes may

be observed in the liver. The mesenteric glands are usually enlarged.

Symptoms.—A case of mild catarrhal ileocolitis begins with fever, diarrhea, and at times vomiting. It cannot be distinguished from other forms of diarrhea until mucus and blood make their appearance in the stools. Instead of the symptoms abating after the intestinal tract has been emptied, there is a persistence of the same and the child continues to have small, frequent odorless dejecta consisting in the main of mucus. The abdomen is not distended as in fermental diarrhea but may be soft and sunken.

As the case advances the stools become more irregular. Some are large, containing mucus, undigested food particles and serum in abundance while again others are simply a stain of mucus on the diaper as a result of the tenesmus that plays so prominent a role in ileocolitis. On account of this straining there is a strong tendency to the development of prolapsus ani.

The constitutional symptoms are fever of moderate grade, with diurnal variations ranging from 100° to 102° F., although at the onset it may be much higher for a short period; prostration; loss of appetite and in some cases vomiting. The duration of the acute symptoms is about a week. Convalescence is slow and is characterized by a tendency to recurrence of mucus in the stools as soon as we attempt to put the child back on its customary diet.

Severe catarrhal ileocolitis presents symptoms that have much in common with dysentery. Constitutional symptoms are pronounced. The fever is high throughout the entire course of the disease and the stools are frequent, are accompanied by painful straining and consist mainly of bloody mucus.

Prostration and nervous symptoms are marked. The child presents the picture of a severe infection—dry, coated tongue; cracked, bleeding lips; apathy or great irritability; anorexia and thirst; prostration. The prognosis is grave; if recovery takes place we may look for a protracted convalescence on

account of superficial ulceration of the gut. The duration is from two to three weeks. Death usually occurs during the second or third week in fatal cases as a result of exhaustion, general sepsis or a secondary bronchopneumonia.

Follicular ulceration is to be suspected in children of weakly constitution who have had repeated attacks of diarrhea or a protracted ileocolitis, and who continue to have a moderate fever and persistent mucus in the stools. The accompanying symptoms are progressive emaciation and failure of strength; anorexia; bed sores; thrush, etc. The duration is long; the condition is practically a sub-acute one. The course is marked by improvement and exacerbation and may be protracted for two to three months. Even after the ulcers have healed there is persistent indigestion and tendency to diarrhea for some time. The characteristic symptoms may be summed up as continued loose movements, four to eight daily, consisting chiefly of greenish mucus; slight fever; emaciation; absence of blood in the stools.

Membraneous Colitis.—This severe and rare form of enteritis may present the most uncharacteristic symptoms; indeed, we may fail to recognize the intestinal condition early in the attack owing to the predominance of the toxemia.

When the onset is abrupt and accompanied by cerebral symptoms it closely simulates meningitis. High fever; convulsions; retraction of the head and abdomen; vomiting and stupor may be present for several days before our attention will be directed to the intestines by the appearance of bloody stools and probably prolapsus ani.

The majority of cases, however, simulate severe catarrhal ileocolitis with the exception that shreds of membrane appear in the stools and may be seen at times upon the rectal mucosa during prolapsus. In all doubtful cases the stools should be carefully washed and strained, as it is difficult to distinguish membrane from mucus when the latter is abundant.

The course is protracted and the prognosis is grave.

The diagnosis rests upon the evidence of severe inflammation of the large intestine, especially of the descending portion. The characteristic symptoms are continued high fever and prostration; frequent, small stools consisting mainly of blood and mucus, with shreds of membrane in the stools. There is tenderness along the entire course of the colon, but particularly along its descending portion. In *typhoid fever* tenderness is only found in the ileo-cecal region and the stools are large, consisting mainly of the contents of the small intestine.

In *meningitis* the bowels are constipated and the cerebral symptoms progress in regular order from day to day. In dysentery they are purely toxic and therefore vary; in fact, they may improve, while the intestinal symptoms increase in severity.

Pain, tenesmus, vomiting and prostration may suggest *intussusception*, but in this condition the onset is abrupt, there is no fever, and an abdominal tumor may be demonstrated.

Treatment.—The first and most important factor in the treatment of the infectious diarrheas is *prophylaxis*. This begins with the care of the child, especially during hot weather. Overdressing must be avoided and the child should receive an abundance of fresh air. The feeding must be intelligently supervised and all attacks of dyspepsia should receive prompt attention.

The disinfection of the stools is important as not infrequently diarrhea becomes epidemic in a family, or hospital ward.

The supervision of the food is of the greatest importance. Use only fresh, clean milk. Boil the drinking water for the baby. Pasteurization will not make dirty milk wholesome. If chemical changes have occurred in the milk, sterilization will not overcome them. Another important point in prophylaxis is not to wean an infant during the summer. There are times when this becomes necessary, but whenever at all possible we should wait for the advent of cool weather before taking this step.

In the summer no infant should be kept in the city if it is at all possible to take it to the country or seashore.

Bathing is most essential during hot weather. The cool or tepid bath is absolutely necessary when fever is present; this may be given three to four times a day.

As a matter of precaution it is safer to pasteurize all milk, even the best obtainable, during the hot summer months. The nipples should be boiled every day and the bottles filled with hot water and washing soda as soon as emptied. Before they are refilled they should be cleansed with a bottle-brush and thoroughly rinsed with plain hot water.

In hot weather infants should receive an ounce or two of water, previously boiled and then cooled, several times daily.

An important point to bear in mind is that during hot weather an infant cannot take as strong a milk mixture as during cold weather. It will usually take the same quantity because it is thirsty, but unless we cut down the fat and sugar we may set up a severe indigestion. Do not expect an infant to gain steadily during July and August.

When diarrhea develops we must at once make appropriate changes in the feeding. In a breast-fed infant, in the absence of fever and vomiting, we may for a day or two continue with the breast milk, but lengthen the interval between nursings. Should the condition not improve it will be wise to alternate the breast with a bottle of barley-water and thus give the digestive organs a rest.

The reason why milk is discontinued in the diarrhea of infants is because it acts as a good culture medium for the intestinal bacteria. According to Finkelstein, it is especially the lactose and the salts of the whey which are the offending elements. On the other hand, the casein of the milk, largely on account of its high calcium content, seems to exert an inhibitory effect upon the fermentative changes present in the gut in so many of the cases of summer diarrhea. Albumin-milk, therefore, frequently proves to be a most successful diet for infantile diarrhea.

The chief need of the organism during the first twenty-four

hours of the illness is water and not food. This should be plentifully supplied in the form of plain boiled water, barley-water or weak tea. If the infant refuses to take water freely it may be sweetened by the addition of a grain of saccharine to the pint. The bowels should be thoroughly flushed with a high enema of normal salt solution and this may be repeated according to indications.

On the second day lamb or chicken broth, cooked with rice or barley, strained and then cooled in order to remove the fat, may be given in quantities slightly less than the infant was taking before its illness and at four hour intervals, water being given between feedings.

On the third day, if the fever has subsided and all curds have disappeared from the stools we may cautiously begin with a milk preparation, preferably "Eiweissmilch," or albumin milk. The beneficial action of this food in diarrhea is largely due to its high protein and calcium content, which favors the formation of soap stools in the intestine. Peristalsis is thereby checked and diarrhea overcome. The presence of buttermilk in this food may also exert a beneficial effect through the antagonistic action of the lactic acid bacilli to the gas bacillus and colon group.

It is not always possible to employ "Eisweissmilch" because of the skill and care required in its preparation. As a routine measure I have found the use of dilute skimmed milk preparations highly practicable and generally successful. We should begin with one part skimmed milk to three parts barley-water, rice water or arrowroot-water, mixed and boiled for five minutes.

The proportion of milk may be cautiously increased until two parts of milk to one of diluent is reached. With this food the stools usually become yellowish, salve-like and alkaline in reaction. When this result is attained one of the maltose preparations may be cautiously added to the food. We should also do this when feeding "Eiweissmilch," for without a suffi-

cient amount of carbohydrate in the food the infant soon becomes seriously emaciated. A gradual return to whole milk should be made after the diarrhea and fever have been definitely controlled, but the milk should be boiled for at least several weeks after an attack of enteritis.

In ileocolitis, or dysentery, Kendall recommends the use of lactose on account of its antagonistic action to the dysentery bacillus. Cases of ileocolitis are often starved entirely too long and thus unnecessarily weakened. We should remember that the lower bowel is involved in these cases and that properly modified milk and thin gruels are well digested before they reach the inflamed colon.

In ileocolitis it is common to find several mucous stools in succession followed by a pasty milk stool. Albumin milk or boiled skim milk mixtures are usually well tolerated.

Special Symptoms and Their Management.—*Vomiting* is at times a most troublesome complication, especially in cholera infantum. Lavage of the stomach is the most rational and successful method of treatment to control it. In urgent cases it may be necessary to perform the operation several times a day, and then pour a little food into the stomach before removing the tube. Thin arrowroot-water or albumin-water is best retained under these circumstances. Often the food will be retained better if fed with a teaspoon than when taken from a bottle.

Diarrhea.—In the early stages of an intestinal infection irrigation of the bowel is beneficial. The gut seldom empties itself completely for which reason the diarrhea and toxic symptoms persist. It is true, the irrigating fluid does not reach beyond the ileo-cecal valve, but, as the colon receives the brunt of the attack in most instances, we help the case materially by cleansing this part of the gut. Besides, irrigation stimulates peristalsis, and thus aids in emptying the portion of gut above this point.

Foul stools and excessive mucus call for irrigation, but we

must discontinue as soon as the improvement sets in as a diarrhea may be prolonged by too much mechanical interference.

Tenesmus may be relieved by injections of olive oil.

High fever is best controlled with baths. Infants may be tubbed two or three times daily in water gradually reduced from 90° F. to 80° F., while older children are more conveniently sponged with cool water and alcohol. Irrigation of the colon tends to control the pyrexia. The child should be kept in the open air as much as possible.

Collapse calls for stimulation. Brandy should only be used when needed, and not given continually during the illness. In grave cases a hypodermic injection of camphorated oil may be necessary. Five minims may be given to an infant one year old. Camphor suits this condition admirably, and it is best given hypodermically, as it may otherwise irritate the stomach. Artificial heat should also be applied when the body surface becomes cold or the temperature subnormal. When the infant has been rapidly depleted by frequent watery stools hypodermoclysis may be resorted to. From fifty to one hundred cubic centimetres of normal salt solution may be injected subcutaneously with a large sized Luer Syringe. The intraperitoneal administration of normal salt solution is more effective than the subcutaneous method and is perfectly safe when properly performed. In cases of *acidosis* Howland recommends fifty cubic centimetres of a four per cent solution of sodium bicarbonate subcutaneously or intravenously.

Remedies.—The diarrhea accompanying teething is especially benefited by *chamomilla*. In acute gastro-intestinal intoxication *belladonna* appears most frequently indicated on account of the predominance of fever and nervous symptoms. Even in the later stages, when the bowel symptoms become more prominent, *belladonna* is of value as long as fever and toxemia are present.

In the ordinary case of fermental diarrhea and ileocolitis *podophyllum* 3 x is a good routine remedy. *Mercurius*

vivus 3 x trit. may be given later, if ulceration takes place. This is indicated by the continuance of the diarrhea, moderate fever and a persistence of mucus in the stools. In the dysenteric type of colitis, *mercurius corrosivus* 6 x is the chief remedy.

Arsenicum, *ipecac* and *veratrum album* are the most useful remedies in cholera infantum. *Veratrum* is Jousset's favorite. *Iris versicolor* will check the vomiting speedily, but leaves the bowels untouched according to Richard Hughes. *Arsenic* and *veratrum* are often difficult to differentiate, especially in the beginning of the case. Under these circumstances they may be given in alternation. I have often found that when one of the apparently indicated remedies fails to act, prompt improvement will follow on giving a constitutional remedy in alternation with the same. Among these *calc. phos.* stands foremost.

Acon.—In the beginning; high fever and restlessness; green mucus in the stools.

Apis.—Cerebral symptoms; suppression of urine; coma, with hot head, dry skin; shrill cry; hydrocephaloid state.

Arsen.—Watery stools, with vomiting and collapse; stools offensive, first greenish, later becoming dark or brownish, and acrid; also small mucous stools with tenesmus. Child nurses often, but takes only a small quantity at one time. Mainly differentiated from *veratrum album* by presence of greater prostration and toxemia.

Bell.—Green stools, abdomen distended and sensitive; face red, high fever. Where inflammatory symptoms are pronounced *belladonna* is the most important remedy especially when brain symptoms develop.

Bry.—Diarrhea brought on by change of weather; stools brownish, worse from moving about; great thirst for large quantities of water.

Calc. carb.—Stools light in color, sour odor; sour vomiting; dentition; rachitic tendency; belly large.

Calc. phos.—Child looks old, under-developed; stools greenish,

thin and offensive; history of tardy dentition; belly flabby. A most valuable tonic both during the disease and in convalescence.

Camphor.—Sudden appearance of choleraic symptoms; great prostration and collapse, body cold yet child will not remain covered.

Cham.—Stools green, with white particles, looking like "spinach and chopped eggs;" fretful; one cheek red, the other pale; child wants to be carried about.

Colocynth.—Painful cases; pressure gives relief.

Croton tiglium is of clinical value in gastroenteric infections where the stools are profuse and watery and of a yellow color. The mother will tell you that every time the child takes the bottle it has a bowel movement, drinking apparently exciting peristalsis and bringing on a stool. It is distinguished from *podophyllum* by a less amount of gas and mucus and absence of straining.

Cupr. ars.—Painful cases; choleraic and convulsive symptoms predominate.

Ipecac.—Nausea and vomiting; stools green as grass, or like yeast. Early stages of cholera infantum.

Mag. sulph.—Dr. Frank H. Pritchard (Hahnemannian Monthly, Nov., 1900) reports favorable results from the use of a weak solution of the *Sulphate of Magnesia* in the summer diarrhea of children. His dosage is one-half to one grain dissolved in a teaspoonful of water. The indications calling for it are copious, watery stools, deficient in bile. He noted that as soon as the remedy had begun to act favorably the stools became bile-tinged.

Mercurius.—A predominance of mucus and involvement of the lower colon calls for *mercury*. The *bichloride* is often preferable to the metal in dysentery; *mercurius dulcis* has grass-green stools. The "never-get-done" feeling of *mercurius sol.* is very characteristic, while the *bichloride* has tenesmus of the bladder as well as of the rectum, and is the chief remedy in true dysentery.

Podophyllum.—Painless, yellowish or greenish, watery diarrhea; prolapsus ani. The stool is expelled with a spluttering sound due to the presence of gas in the bowels. The buttox becomes excoriated. *Podophyllum* is the best routine remedy in acute enteritis.

Sulphur.—Excoriating stools, worse mornings; marantic cases. The child is peevish and has a voracious appetite. The lips are abnormally red and the anus is excoriated. Exudative diathesis; eczema.

Veratr. alb.—Vomiting and purging, the latter most prominent; motion aggravates all symptoms, cold sweat on forehead. There is less prostration and thirst than with *arsenicum*, less restlessness and usually more pain, and when any doubt exists as to a choice between the two, *veratrum* should receive the preference early in the case. *Arsenicum* is indicated in the later stages when exhaustion and prostration have become the prominent symptoms.

CONSTIPATION.

Constipation is one of the commonest and most troublesome conditions encountered in infancy. As a rule it is of no serious import, being mainly dietetic in origin. It may, however, result from atony of the intestines and abdominal walls and is, therefore, frequently associated with rickets. In some instances there is an anatomic basis; this may be an elongated sigmoid, prolapse of the colon or congenital dilatation and hypertrophy of the colon (Hirschsprung's disease). Fissure of the anus, by causing a reflex spasm of the sphincter ani, may also be a cause of stubborn constipation.

A normal nursing infant usually has from three to four bowel movements daily, while an artificially fed infant generally has but one or two. In the former instance the stools are thin and contain small, soft curds of fat, although they are at times pasty, while in the artificially fed infant the stool is of firmer consistency and more homogeneous. After the infant is weaned and other foods than milk are added to the diet there is

usually one bowel movement daily, approaching the type of stool seen in adults.

In the majority of instances some dietetic error will be found responsible for the condition, although it should not be forgotten that infants are naturally predisposed to constipation on account of the relatively great length of the intestinal tract and the exaggerated curve of the sigmoid flexure. The musculature of the intestines is relatively feeble, and for this reason long continued impaction of the gut with fecal residue and overdistention from fermentative processes may lead to permanent dilatation of the colon. It is a mistake to believe that fecal impaction is uncommon in young children, and whenever abdominal symptoms are encountered this is one of the first conditions to be looked for.

Again, habit must also be taken into consideration, for it plays a prominent role in the etiology of constipation in children as well as in adults. A great many cases are the direct result of bad training; it is surprising how early some infants can be taught to have regular bowel movements.

An important point to bear in mind is that the infant may appear to be constipated when in reality it is not getting sufficient food or the same is so deficient in solids that there is not enough fecal residue in the intestinal tract to produce the usual number of evacuations.

It is generally taught that deficiency in fat in the food is the chief cause of constipation in infants, and while this is true to a certain extent, nevertheless much harm has been done by the indiscriminate application of this principle. There is no doubt that the increase of fat in a milk formula beyond a certain percentage may not only aggravate the constipation, but may also induce a train of serious general symptoms resulting from disturbed metabolism and fat dyspepsia. An increase in carbohydrates is more important than an increase in the fat, excepting in cases which have been on a diet with distinctly low fat percentage. It is well to remember that the percentage of fat in

a formula should never be increased beyond 4 per cent, and that, as a rule, 3 per cent of fat is sufficient for the infant's requirements.

The substitution of maltose for lactose in the food will also act beneficially in many instances in relieving constipation. Oatmeal water, used as a diluent for the milk, exerts a laxative effect. A drink of sugar water between feedings will often give good results.

In the case of older children we have more latitude in the regulation of the diet. Fruit and a glass of water before breakfast should be insisted upon. The amount of meat should be restricted, and the eating of cereals, vegetables and fruits either raw or stewed, should be urged. Graham bread should be substituted for wheat bread. Agar may be given with a cereal and is more efficacious than bran. A useful adjuvant in the treatment of these cases is a dessert-spoonful of olive oil mixed with an ounce of unfermented grape juice taken twice daily, one hour after meals.

Local conditions, such as fissures of the anus and polypi, must be looked for. The systematic use of *enemata* is of decided value in habitual constipation, and their employment is especially indicated when the stools are large and hard. A gluten *suppository* may also prove valuable in establishing the habit of evacuating the bowels at a regular time each day.

Massage is of value in infants to stimulate peristalsis and assist in the dislodgment of fecal accumulations.

The remedies most frequently useful are *bryonia*, *hydrastis*, *nux vomica* and *sulphur*. Other remedies which may be called for upon special indications are *alumina*, *calc. carb.*, *graphites*, *lycopodium*, *mercurius vivus*, *opium*, *phosphorus*. If it becomes necessary to resort to physiological measures we should avoid the use of drugs which are either harmful or which only aggravate the condition.

Castor oil should only be used in acute conditions in which a rapid emptying of the bowels is imperative.

Hydrated magnesia, which is marketed as “milk of magnesia,” may be added to the milk in place of lime water, and thus serve the double purpose of an alkali and mild laxative. One drachm added to the twenty-four hours’ amount of food will usually be found sufficient. Older children may take a teaspoonful in water at bedtime.

The general and indiscriminate use of the popular syrups (proprietary) which contain senna and Rochelle salts, is to be discouraged, although *senna* is the least harmful of the various laxatives.

Ox-gall is a cholagogue, and may be given in one grain doses in cases characterized by deficient biliary secretion.

Bryonia.—Stools large and dry, as if burnt.

Graphites.—Stool consists of small balls bound together by mucus. Fissure ani; eczema ani; fat babies with skin eruptions.

Hydrastis.—Constipation due to atonic dyspepsia and portal congestion; mucous colitis; catarrhal affections in general; loss of appetite. “Constipation after purgative medicines”—(Goodno).

Nux vomica.—The child strains and grunts but passes little or no stool; the abdomen is distended and hernia is apt to result from the constant straining.

Sulphur.—Habitual constipation with general malnutrition; anus sore after stool; prolapsus ani and hemorrhoids; alternate constipation and diarrhea.

INTUSSUSCEPTION.

Intussusception is most frequently seen in infancy, and has been found to occur oftener in boys than in girls. It is probably due to increased peristalsis and occurs, as a rule, in poorly nourished infants during an acute intestinal disturbance.

The condition is one of invagination of one portion of the intestine into another, most frequently the lower end of the ileum, together with the cecum being invaginated into the colon. The invaginated portion produces a sausage-shaped tumor in

the region of the cecum or transverse colon, often advancing over into the left lower quadrant of the abdomen. It may at times be felt in the rectum, even protruding therefrom.

The onset is usually sudden, the symptoms being colicky pains, with vomiting and straining at stool. The lower bowel soon becomes emptied of its fecal contents, after which passages of blood and mucus make their appearance. The vomiting becomes stercoraceous unless the obstruction is relieved, and the patient dies in collapse.

Spontaneous reduction or sloughing of the invaginated portion of the gut, and successful union with restoration of the lumen of the canal may occur in exceptional instances.

The *prognosis* is grave unless the intussusception can be reduced within a reasonable time of its occurrence. Gibson (*N. Y. Medical Record, July 17, 1897*) estimates the mortality as 53 per cent from a collection of 249 cases. Early operation is indicated.

Diagnosis.—The majority of cases of intestinal obstruction occurring in infancy are due to intussusception. Symptoms of obstruction, therefore together with the presence of the sausage shaped tumor in the abdominal cavity and in the rectum, bloody stools and active movements of the intestinal coils above the seat of obstruction and projectile vomiting and collapse are the symptoms upon which the diagnosis should readily be made.

APPENDICITIS.

Appendicitis is seldom seen as early as intussusception, only exceptionally occurring during infancy, and rarely before the fourth year. The causes are the same as in adults. *Appendicular colic* is more common in children, however, than in adults, owing to the more patulous state of the opening of the appendix into the cecum, permitting the entrance of fecal concretions (*stercoraceous appendicitis*).

The *catarrhal variety* is characterized by its mild course and absence of complications. It is usually of the *chronic recurring variety*.

The *perforative variety* is accompanied by fever, pain, local tenderness and rigidity and a leucocytosis. Unless prompt surgical treatment is instituted there is always the danger of perforation and septic peritonitis occurring.

The clinical features of appendicitis are very characteristic, and are tersely and clearly described by Van Lennep ("Appendicitis," *Trans. of the American Institute of Hom.*, 1897):

"There is the history of improper eating, or perhaps exposure to cold, associated with the menstrual period in the female; occasionally over-exertion, particularly in the sedentary, or possibly a direct traumatism. Then, what have been aptly termed the cardinal symptoms: (1) *Pain*, at first peri-umbilical or diffuse, but soon referred to the right iliac fossa, unless the appendix points elsewhere. (2) *Tenderness*, almost always present at the junction of the organ with the cecum (McBurney's Point); sometimes associated with other sore spots corresponding with distal lesions or their products. (3) *Muscular rigidity*, to corroborate tenderness, which may vary from a local or general board-like stiffness to an indistinct, circumscribed muscular tension, or a barely appreciable difference between the two recti at their costal margin. Besides this three-legged stool, as Hering would have termed these cardinal symptoms, are the well known concomitants: Sudden onset, the coated, flabby and indented tongue; the vomiting, which, when present, is from an overloaded or rebellious stomach; constipation, sometimes preceded by an irritative diarrhea; distention, usually local in the early 'tympanitic tumor,' due to atony of the cecum from an irritated appendix; and, lastly, as might be expected, a moderate temperature rise and pulse acceleration."

Diagnosis.—With the presence of the above symptoms the diagnosis of appendicitis is not difficult. From intussusception it is differentiated by the absence of projectile or stercoraceous vomiting, bloody stools and intestinal tumor. Furthermore, in intussusception there are active movements of the intestines, while in appendicitis "actively-moving intestinal coils are not

seen or felt and gurgling is scanty or absent" (Van Lennep). Referred pain in the right iliac fossa is not uncommon in pneumonia of the right base in childhood, due to an associated pleurisy. A routine examination of the chest should, therefore, be made in all cases of suspected appendicitis in children.

Treatment.—The majority of cases of intussusception and appendicitis are first seen by the general practitioner. It is his duty, therefore, to be thoroughly familiar with the symptoms and diagnostic signs of these conditions so that he may be able to recognize them and make an early diagnosis. If the surgeon is called in too late, no matter how skillful he may be, he can no longer do justice to either himself or to the patient. The responsibility, therefore rests mainly with the family physician who is the first to see the case.

In regard to surgical treatment Van Lennep says: "My working-plan regarding operation is about as follows: In a severe attack, characterized by sudden onset, and particularly by intense cardinal symptoms, with or without corresponding concomitants, operation should be undertaken at once. In a milder seizure, the more common form, recovery may be looked for, with the hope of an interval operation. In deciding the question of persistence in such cases I have come to rely more than ever on the twenty-four hour limit, and I believe that whenever doubt or would-be conservatism has induced me to delay, I have had cause to regret the inaction. Benign cases will show signs of improvement within twenty-four hours, while unfavorable cases, requiring operation, usually grow worse during this time, and become dangerous from the possibility of perforation with septic infection of the peritoneal cavity."

It is best to discontinue all feeding by mouth and resort to enteroclysis, after a preliminary enema. An ice bag placed over the region of the appendix adds to the patient's comfort. Morphine should not be used as it masks the symptoms of the case. One of the following remedies may be indicated:

Nux vomica.—This is the most important remedy in the early

stage, indicated by coated tongue, nausea and vomiting, colicky pains in abdomen, constipation with urging to stool, abdomen tender and bloated.

Belladonna.—Intense pain and sensitiveness in the right ileo-cecal region, cannot bear the weight of the bedclothes or to be touched. There is high fever, flushed face, vomiting, patient lying motionless on back with right leg drawn up.

Bryonia.—Inflammatory stage. *Bryonia* covers the pathological condition more closely than any other remedy, and its cardinal symptom, pain aggravated by motion, together with inflammatory fever, thirst and constipation, are almost invariably present.

Hepar sulph.—When suppuration has set in.

Mercurius sol.—Painful tumefaction in right ileo-cecal region; tongue broad and flabby, showing imprint of teeth; constipation; fever, worse during night, with sweat, which gives no relief.

INTESTINAL PARASITES.

The parasites which may be found infesting the intestinal tract of children are two round worms, the *oxyuris vermicularis* and *ascaris lumbricoides*, and two tape worms, *tenia saginata* and *tenia solium*.

There are no characteristic symptoms upon which intestinal parasites can be positively diagnosed. In the case of the *oxyuris*, itching about the anus is commonly present and should arouse suspicion of the presence of seat worms. The symptoms attributed to the *ascaris*, or "stomach-worm" are, however, often produced by other conditions such as intestinal indigestion or they are of neurotic origin. A positive diagnosis of the presence of these parasites can therefore only be made by finding the parasite or its ova in the stools. Many children who have worms, present no symptoms while others who grit their teeth at night and have digestive disturbances or febrile attacks of intestinal origin show no evidence of worms in their stools.

Symptoms.—The characteristic disturbances induced by the

oxyuris vermicularis, or seat worm, are pruritus ani at bedtime, recurring regularly each night, in some cases even pains in the rectum; enuresis, and in male children erections with consequent masturbation. The worms also migrate into the vagina in females, inducing leucorrhea and masturbation. They are found in the feces, and can be detected at night emerging from the anus.

The *ascaris lumbricoides* should be suspected when there are attacks of colicky pains; intestinal catarrh with loose stools or mucus in the feces; nausea and vomiting not due to disordered stomach; irregular appetite; pale countenance with dark circles under the eyes; dilated pupils; itching of the nose; gritting of the teeth; restless sleep with starting, and atypical febrile disturbances. All of these symptoms, however, may be induced by other conditions, and these should be excluded before a diagnosis of worms is made.

Dr. La Fuente (*Presse Medical*) considers attacks of intestinal colic coming on suddenly, seizing the child usually at play, and confined to one part of the abdomen, and bilateral narrowing of the field of vision as pathognomonic signs of ascarides. During the attacks of colic the abdomen is quite sensitive to palpation at the seat of the pain. These symptoms can undoubtedly result from worms but we must be on our guard and not mistake an attack of appendicitis for one of worm colic. It should also be remembered that the round worm sometimes migrates into the appendix and thus sets up a genuine attack of appendicitis.

Tape-worms are far less commonly encountered in children than the seat worm and round worm. They are usually unsuspected until segments of the worms are passed, although tape-worms may produce marked anemia. In every case of severe anemia the stools should be examined for parasites.

Morphology.—The *oxyuris vermicularis*, also known as the thread-worm and seat-worm, is a small, whitish, thread-like parasite attaining a length of 10 m.m. in the case of the female, and 4 m.m. in the male. The female has an acuminate tail,

while the male is blunt. They infest the lower ileum and upper colon, often in great numbers. The females prefer the cecum and the colon, and, when mature and egg-bearing, migrate into the colon and rectum to deposit their eggs, whence they also creep out of the anus at night. The eggs are oval, flat on one side and rounded on the other, and exceedingly small. Before they can develop they must first enter the stomach of some host, and it is quite likely that a child often reinfects itself by swallowing the eggs from its own colony of parasites.

Ascaris lumbricoides.—This is the common round worm, being cylindrical in shape, with tapering extremities and light reddish-brown in color. The female may attain a length of fifteen inches; the male eight to ten inches. The eggs are larger than those of the oxyuris, and possess a double shell, the contents being dark and granular. They measure about $\frac{1}{340}$ inch in length. The mature female sheds enormous numbers of these ova. The life history of the ascarides is not fully understood. They infest mainly the small intestines, although they may be found at any point in the alimentary tract, sometimes even being vomited, and in rare instances inducing death by creeping into the ductus communis choledochus or into the larynx.

Tenia saginata.—This tape-worm is derived from beef, and is perhaps the most common variety in this country. It has a square head, with four suckers, but no hooks. It may attain a great length, and the segments are very numerous, and longer than broad. The life-history of the worm is as follows: After the eggs are discharged into the intestinal tract by the mature segments, they reach the alimentary tract of oxen grazing on pastures where the infected stools have been deposited. Here the embryos are liberated and find their way into the muscular tissue throughout the body, and sometimes into various organs, where they become converted into the cysticercus, or larval form. If this cysticercus is eaten with raw or insufficiently-cooked meat, the capsule is destroyed by the digestive juices and the contained scolex liberated, which attaches itself to the mucous

membrane of the small intestine, where it soon develops into the fully-matured form by segmentation.

Tenia solium, also known as the armed tape-worm, is derived from pork, and differs from *t. saginata* in being equipped with a set of hooks besides two pairs of suckers. This parasite is also much smaller than the other variety and is less frequently encountered in the United States.

Treatment.—In the case of oxyurides the best results are obtained from the use of high enemata of salt water. These must be given every night for several nights in succession. The salt water irrigations may be discontinued for a time and olive oil injections into the rectum substituted, using from three to four ounces of oil and letting the child retain the same over night. Should these measures not bring relief, a purgative, followed by a high enema of infusion of quassia may be employed.

Ascarides are best gotten rid of by the administration of *santonin*, half a grain after meals for three or four doses followed by a purgative. The ascarides are not killed by the *santonin* but driven into the large intestine whence they are readily expelled by a dose of *castor oil* or *citrate of magnesia*.

For the expulsion of the tape-worm the *oleoresin of male fern* is the most reliable anthelmintic. Fifteen minims should be given after a fast of twelve hours and followed by a saline purgative.

CHAPTER VIII.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Acute peritonitis is rarely seen in childhood. During infancy it is encountered as a manifestation of the newborn (see "Diseases of the Newborn") while in older children it develops secondarily to such conditions as appendicitis, intussusception and empyema. Acute peritonitis may also complicate the acute infectious diseases.

Symptoms.—The onset of peritonitis is characterized by the development of painful abdominal distention with rigidity and tenderness; vomiting, usually of bile and an increase in the fever and rising leucocyte count. Since peritonitis is usually secondary to some other condition the advent of the above symptoms should at once arouse suspicion of a complicating peritonitis.

As the condition progresses peristalsis is abolished; the respirations become shallow and the abdomen does not move with the respirations and the face assumes the "abdominal, or Hippocratic" expression. The eyes are hollow and sunken, the features become pinched, the face is cold, there is a distressed expression and the mind remains clear. The pulse is small and rapid and collapse is impending. The outcome is usually fatal in cases of septic peritonitis.

Treatment.—All feeding by mouth should be discontinued and water should be given by enteroclysis. Hot fomentations may be used to relieve the pain. When vomiting becomes a troublesome symptom lavage should be resorted to.

The remedies useful in the early stages are *acon.*, *bell.* and *bry.* In the latter stages *apis*, *canth.*, *mercurius corr.* and *rhus tox.* are indicated..

Acon.—Sudden onset with chill; hot, dry skin; rapid, hard pulse, with high fever, great restlessness and anxiety. Cutting and darting pains in bowels or burning in the abdomen.

Apis.—Exudation; burning, stinging pains; scanty urine; loud piercing shrieks and cerebral symptoms; pneumococcus peritonitis coexisting with meningitis.

Arnica.—After traumatism, in early stages.

Arsenicum.—Later stages, impending collapse. Great anguish; clammy perspiration; the patient feels cold, and complains of burning pains in abdomen; restless tossing, thirst, obstinate vomiting, distention of the abdomen and cold extremities.

Camphor.—Collapse.

Cantharis.—Intense inflammation, pinched features, rapid, feeble pulse, cutting and burning pains. (Goodno.)

Carbo veg.—Great distention of the abdomen, with paralysis of the bowels. Extremities cold up to the knees; collapse.

Lachesis.—Great hyperaesthesia of the abdomen; complicating gangrenous inflammation of the appendix; loquacious, adynamic fever.

Merc.—When the exudate tends to become purulent, indicated by chilliness, followed by sweat; starting in sleep; cachectic expression; foul breath; emaciation; obstinate vomiting.

Opium.—Paralysis of bowels or antiperistaltic action; incessant vomiting, distention of abdomen, somnolence and stupefaction; warm sweat.

Rhus tox.—The *rhus tox.* patient prefers to lie on his back with the legs drawn up, although the pains make him very restless. There is delirium at night, great prostration, and a brown tongue with red tip.

Sulphur.—To hasten resorption of exudate.

CHRONIC PERITONITIS; TUBERCULOUS PERITONITIS.

Chronic peritonitis is usually tuberculous. Fetal peritonitis may be of syphilitic origin. Tuberculous peritonitis is usually

secondary to intestinal tuberculosis, infection taking place from caseating mesenteric glands; primarily it may develop as the acute miliary type or as a part of an acute miliary tuberculosis. Abdominal tuberculosis in children is usually due to the bovine type of tubercle bacillus.

Symptoms.—The following clinical types of tuberculous peritonitis are encountered:

The *acute miliary type* which presents the symptoms of acute peritonitis and is rapidly fatal.

The *ascitic type* which may be idiopathic or tuberculous. It is accompanied by an exudate of serum, or a purulent fluid in case of mixed infection.

In the *adhesive type* there is a matting together of the intestines by bands of plastic exudate. Caseous masses in the mesentery and in the organized exudate, and a firm mass containing coils of intestine, difficult to entangle after the abdomen has been opened, result from the tuberculous process. Obstruction of the bowel finally occurs. The *fibro-plastic* variety presents adhesions together with fibrinous exudation, which may undergo caseous degeneration and induce ulceration into neighboring organs. Fecal fistulæ forming in the neighborhood of the umbilicus are not uncommon in these cases.

The *symptoms* of tuberculous peritonitis are progressive loss of weight with moderate fever; anemia; diarrhea which is either persistent or recurring and abdominal distention. The diarrhea usually precedes the development of peritonitis and is due to intestinal tuberculosis.

The *course* is slow and may terminate in spontaneous recovery, especially in the fibro-plastic type, although death usually results from extensive adhesions of the abdominal viscera. The ascitic form frequently recovers under surgical treatment, but relapses and general infection are always to be feared.

Diagnosis.—The presence of fluid in the abdominal cavity, together with evidence of a thickened omentum and nodular

masses in the region of the small intestines should suggest the diagnosis of tuberculous peritonitis especially if other evidences of tuberculosis are present. These latter are interscapular dulness, fever and emaciation and a positive von Pirquet test. A preceding chronic diarrhea should suggest intestinal tuberculosis. The abdomen is uniformly distended and fluctuating and shifting dulness in the flanks can be elicited in ascites without adhesions. Ridges and irregular contour of the abdomen suggest adhesions while stationary dulness speaks for sacculated fluid. In the adhesive variety an irregular tumor can usually be palpated. Chronic obstruction of the bowels gradually develops when the adhesions constrict the gut.

In the presence of marked ascites, hepatic disease must be excluded before a diagnosis of peritonitis can be made with certainty. In peritonitis the fluid contains more albumin and is of a higher specific gravity than in peritoneal effusions of circulatory or renal origin. There is also a high mononuclear cell count. Cirrhosis of the liver does not occur in childhood but pseudo-cirrhosis accompanying adhesive pericarditis (polyserositis) is not uncommon in children and must be excluded (see "Diseases of the Heart").

Treatment.—Owing to the favorable results obtained by laparotomy, every doubtful case should receive the benefit of an exploratory incision. The ascitic variety is the one especially benefited by laparotomy and evacuation of the fluid; in the others it is of doubtful value. Sutherland (*Archives of Pediatrics*, Feb., 1903) is not in favor of surgical intervention since in forty-one cases observed by him the results of medical treatment of all varieties were much better (70 per cent recovered) than of surgical treatment (50 per cent recovered, 50 per cent died). The usual regime of diet and fresh air should be carried out. *Tuberculin* in minute doses, either internally or subcutaneously should be tried. Other remedies to be considered are *iodine*, the *iodide of arsenic* and *calcerea carb.*

CHAPTER IX.

DISEASES OF THE RESPIRATORY TRACT.

SPASM OF THE GLOTTIS.

Spasm of the glottis, or *laryngismus stridulus*, is one of the chief clinical manifestations of the *spasmophilic diathesis*. It should not be confounded with the respiratory difficulty resulting from an enlarged thymus gland, the so-called thymic asthma of Kopp, which is a symptom of the *lymphatic diathesis*, or *status lymphaticus*.

Laryngismus stridulus is most frequently encountered during the first year; it is rare in older children and more common in boys than in girls. Symptoms of rickets are usually associated.

An attack is usually brought on by crying or by fright. Sometimes it comes on without any apparent cause. The child suddenly holds its breath and then makes ineffectual efforts to inspire which is made impossible by the spasmodic contraction of the vocal cords. The features become cyanotic and temporary asphyxia results. This may last until partial unconsciousness and complete relaxation ensue, after which inspiratory efforts, accompanied by a crowing sound, are made. Consciousness returns, the crowing sounds disappear and the child is again normal. Death from cardiac failure may occur during one of these attacks (*cardiac tetany*).

Congenital stridor of infants is a condition which should be mentioned in connection with glottic spasm. Its true nature is not fully understood. In some cases disturbed innervation of the laryngeal muscles is perhaps present while in other cases there is a congenital malformation of the glottis.

Treatment.—Since laryngismus stridulus is only a symptom, treatment must be directed toward improving the underlying constitutional disturbance. The infant should therefore be

thoroughly examined for evidences of spasmophilia and rickets and treated accordingly.

During an attack of laryngeal spasm the child should be sprinkled with cold water and the index finger introduced into the throat to raise up the epiglottis, as in performing intubation. A quick acting stimulant like camphor, given hypodermically may be required. *Belladonna* and *magnesia phos.* may be given to lessen the tendency to a return of the spasm. The diet and constitutional treatment is that of rickets.

ACUTE CATARRHAL LARYNGITIS; SPASMODIC CROUP.

This form of croup, which must be distinguished from *true croup*, or *membranous croup* (*laryngeal diphtheria*), is a common affection of childhood, being a catarrhal inflammation of the mucous membrane of the larynx associated with spasm of the interior laryngeal muscles.

The anatomical and physiological peculiarities of the larynx and nervous system of young children, namely, the relative smallness of the larynx and rima glottidis, the great vascularity of its lining mucous membrane and the heightened reflex excitability of its nerve-supply, offer a ready explanation for the frequency of this malady during childhood and the spasmodic and paroxysmal character of the symptoms.

Etiology.—The chief exciting causes are exposure to cold, draughts or wet weather; acute indigestion and direct irritation, such as the inhalation of dust or contaminated atmosphere. Attacks occur more frequently during the winter and early spring than in the milder and dryer season. Male children are more frequently attacked than females, and the age at which it is most likely to occur is between the second and third year. The majority of children who are subject to croup are of a nervous temperament and have enlarged tonsils and adenoids.

Symptoms.—The characteristic feature of spasmodic croup is its paroxysmal nature. The child may have been well during the day, but towards night a ringing metallic cough—sometimes

before retiring, at other times not until the child has been asleep—makes its appearance. Usually the child is aroused from sleep by an attack of cough and choking, as a result of which, it is much agitated and frightened. The breathing is oppressed, inspiration almost impossible, prolonged, and accompanied by a harsh rasping sound, while with expiration there is a ringing, metallic (croupy) cough.

The attack may last from a few minutes to an hour or longer, not, however, in one continued degree of severity. A second milder attack is likely to occur during the same night, and on the following night it may be looked for with all probability. A moderate amount of fever, together with some catarrhal secretion is usually present. The condition rarely results fatally.

A more severe form of *acute laryngitis* is at times encountered, simulating membranous croup. In these cases there is more fever and more severe inflammation of the throat and cultures for the diphtheria bacillus are negative.

Spasmodic croup is differentiated from membranous croup by the absence of constitutional symptoms; absence of dyspnea between the choking attacks; absence of aphonia and of exudate on the tonsils and in the larynx. Membranous croup becomes progressively worse while catarrhal croup is paroxysmal, worse at night and recurring in nature.

Treatment.—During an attack of croup the inhalation of steam from a croup-kettle helps to relieve the cough and difficult breathing. The rooms should be kept warm and warm drinks may be given. A warm pack about the throat will also prove beneficial.

Many remedies have been recommended for croup and an emetic is sometimes given for the purpose of loosening the secretion and overcoming the laryngeal spasm. In my experience, however, the effect obtained by the administration of *ippecac* is disappointing. *Belladonna* 2x and *tartar emetic* 3x trit. given every fifteen minutes alternately during the attack and every hour between attacks give the best results. To avoid

recurrences the child's general condition should be looked into and enlarged tonsils and adenoids removed.

ACUTE BRONCHITIS.

Acute catarrhal bronchitis is one of the commonest ailments of childhood. It may develop primarily or as an extension of an upper respiratory infection, or common "cold." The microorganisms usually encountered in acute bronchitis are the pneumococcus, the staphylococcus aureus and the streptococcus viridans. During epidemics of influenza the influenza bacillus is the predominating organism. Bronchitis also frequently develops secondarily, being a frequent accompaniment of most acute infectious diseases.

Lack of fresh air and sunshine, overdressing, sleeping in a warm bedroom or living in crowded unhygienic quarters are prominent predisposing causes. Anemia and malnutrition also act as predisposing causes. During infancy rickets is an important predisposing cause; in older children adenoids play an important role. Some children exhibit a constitutional predisposition which may be hereditary.

Several varieties of acute bronchitis are to be recognized. The *mildest form* presents an acute catarrhal tracheo-bronchitis, afebrile in its course and unaccompanied by constitutional disturbances. Babinsky prefers to call it *bronchial catarrh* in contradistinction to actual bronchitis. It is very prevalent among infants during the colder months of the year, and seems to be dependent upon atmospheric changes and constitutional predisposition.

Acute febrile bronchitis is infectious in origin, is accompanied by constitutional symptoms, and tends to spread to the finer ramifications of the bronchial tubes and in early infancy may thus result in *capillary bronchitis*. When the process invades the pulmonary parenchyma, a true bronchopneumonia results.

Pseudo-membranous bronchitis, or *fibrinous bronchitis*, is due to the extension of a diphtheritic infection from the larynx into

the bronchi. A chronic form of obscure origin is occasionally encountered.

Pathology.—As in the case of spasmodic croup, a catarrhal inflammation of the bronchial tubes during infancy is of more serious import and accompanied by more suffocative symptoms than a similar condition in adult life. The greater vascularity of the mucous membrane, and the relatively smaller size of the tubes and air-vesicles are responsible for the development of the serious symptoms which may be noted in bronchitis in infancy. In fatal cases the mucous membrane appears swollen, injected, ecchymosed, and covered with mucus and purulent secretion. In the larger tubes the lining membrane alone is affected, while the smaller and finer ones are involved throughout their entire thickness in the inflammatory process. The lungs are usually emphysematous, from dilatation of the air-vesicles and blocking of the capillary tubes with secretion. Areas of atelectasis are also encountered. Dilatation of the bronchi may also result.

Symptoms.—Bronchitis may run a mild or a dangerous course. In the first instance there is slight fever, cough, which at first is dry and irritating in character, later becoming loose and accompanied by rattling of mucus in the larger tubes. Some soreness in the region of the bifurcation of the trachea may be present, but the child evinces no great degree of pain or discomfort, and within a week or less the attack is over.

When the smaller tubes, however, become involved, the case presents an entirely different aspect. There is rapid breathing, marked dyspnea, imperfect aeration of the blood and enfeebled circulation, higher fever (103° to 104° F. or over), and auscultation of the chest reveals the presence of sibilant rales and fine, moist rales in the bronchi. The child becomes exhausted from incessant coughing and being unable to expectorate the mucus which accumulates in the bronchi, it shows evidence of a gradually increasing asphyxia. It becomes dull and apathetic, even comatose, the pulse rapid and thready or imperceptible, and death, sometimes preceded by convulsions, terminates the scene.

This severe type, described as *capillary bronchitis*, is, strictly speaking, a bronchopneumonic process, and it is impossible to draw a sharp line between an acute spreading bronchitis and pneumonia. As stated above, the pulmonary parenchyma soon shares in the inflammatory process thus accounting for the serious symptoms observed.

Diagnosis.—In bronchitis the percussion-note is not altered. In mild cases there are at first dry rales, followed by large, moist rales, with here and there a sibilant rale, all best heard posteriorly. In the second variety *subcrepitant* and sibilant rales, general in distribution, with large moist and dry rales in the large tubes and trachea, and areas of dullness, with diminished respiratory murmur, indicating collapse of air-cells, may be elicited. Hyper-resonance, resulting from vicarious emphysema, is difficult to recognize in children, as the normal percussion-note is in itself highly resonant.

Sufficient dilatation of some of the bronchi (*bronchiectasis*) to produce physical signs may result. In such cases bronchial breathing may be heard over the dilated bronchus and a tympanitic note can be elicited by percussion. The sputum is purulent and separates into a purulent sediment superimposed by a fluid and frothy layer.

Treatment.—In mild cases of bronchitis it is often advisable to keep the child out of doors as much as possible, instead of confining it to the house. The predisposition to bronchitis may be overcome by cold sponging, an out of door life and the correction of any local or general disturbances which may act as predisposing causes.

Severe cases of bronchitis should receive all the care and attention accorded a case of pneumonia.

The most frequently indicated remedies are *aconite*, *bella-donna*, *bryonia*, *ferrum phos.*, *hepar sulph.*, *ippecac*, *mercurius*, *pulsatilla* and *tartar emetic*. These remedies must be differentiated according to their characteristic symptoms as given below.

Aconite, *belladonna*, *bryonia*, *ferrum phos.* and *mercurius* are indicated in the early stages of the disease while *hepar*, *pulsatilla* and *tartar emetic* are indicated in the later stages.

Aconite has high fever, dry skin, absence of chilly feelings as in *mercurius*, absence of burning heat of the skin, as in *belladonna*, which has a dry, distressing, paroxysmal cough, usually worse towards evening. *Belladonna* is looked upon by some as a specific, it is not necessary, however, to administer it in physiological doses.

The greatest usefulness for *bryonia* is to loosen the cough when the same remains hard, painful and non-productive and is accompanied by soreness of the abdominal muscles. The child is very thirsty, irritable and does not want to be moved. *scilla* is indicated by painful cough; it is, however, a more severe type than *bryonia*, there being cyanosis and failing circulation, owing to extension of the process into the finer tubes.

Cham. suits mild cases of tracheo-bronchitis in the early stages; the cough is excited by attempting to use the voice, and the child is fretful and cross.

Ferrum phos.—Often preferable to *aconite* in cases characterized by hoarseness and dyspnea from the beginning, with rapid progress, soon assuming the capillary variety. The cough is short and dry, often paroxysmal, and when expectoration appears it is streaked with bright blood.

Mercurius.—"Mercurius corresponds with the whole course of a severe attack of bronchitis, even better than *belladonna*. There is violent fever, the temperature is very high, there is a great disposition to sweat without obtaining any relief from it; in contradistinction to *belladonna* there is a constant alternation of chills and heat, with a remarkable sensitiveness to the most trifling changes of temperature" (*Baehr, Science of Therapeutics*). Tongue thickly coated yellow; diarrhea; cough dry, worse evening until midnight; dyspnea; expectoration tenacious.

As the cough becomes loose, *hepar sulph.*, *ipecac*, *pulsatilla* and *tartar emetic* should be differentiated.

CHRONIC BRONCHITIS.

Chronic bronchitis is less common in children than in adults as the reparative processes are more active during childhood and there is less tendency for an acute process, even when there are frequent recurrences, to become chronic. Bronchitis may persist for a long time after an attack of whooping-cough or measles. It may be secondary to Bright's disease or organic heart disease or be a manifestation of tuberculosis.

The important *pathological changes* are thickening of the mucous membrane, with areas of superficial ulceration, weakening and irregular dilatation of the bronchial tubes, and more or less extensive emphysema.

The important *symptoms* are cough and expectoration, and, notwithstanding the long continuance of these symptoms, the general health rarely suffers to a marked degree. Naturally, these children are not up to the normal standard of health, as the etiology of the affection indicates; however, there is not the gradual loss of weight and strength observed in a tuberculous condition. Children with chronic bronchitis usually also have asthmatic manifestations.

The cough is loose, usually paroxysmal, and may become dry and teasing at times. It is generally worse in the morning, and the expectoration of large quantities of offensive muco-pus on rising, associated with localized coarse, moist rales, is strongly indicative of *bronchiectasis*.

The *course* is a slow one and cases may be apparently cured in the summer only to have a relapse during the winter. Nevertheless, the *prognosis* is favorable as a rule, provided that the child can receive the proper hygienic and medical care.

Treatment.—An equable, moderately warm and dry climate is desirable; the mountainous pine regions are especially beneficial. Tonic treatment must be instituted in all cases—baths, fresh air, exercise and a highly-nutritious diet being the essentials.

The mouth, nose and throat must be carefully inspected for evidences of focal infection. Septal deflections, spurs and polpi are frequent sources of irritation but more commonly adenoids and enlarged tonsils will be found. Enlargement of the lingual tonsil is often responsible for persistent winter coughs and should be looked for.

Hepar sulph.—I have found this remedy of especial benefit for the paroxysmal cough coming on at night. A powder of the third decimal trituration will usually relieve the attacks with astonishing promptness.

Pulsatilla is indispensable for the loose cough with profuse easy expectoration of yellowish or yellowish-green muco-pus, having a tendency to become tighter and more troublesome at night. This remedy acts very satisfactorily with *hepar*, and I frequently employ it during the day, giving a dose of *hepar* at night.

Lycopodium is particularly useful for the recurrent type of bronchitis, in which the patient is seldom free from a troublesome cough, "catching cold" on the slightest provocation. "Cough dry, day and night, in feeble, emaciated boys."—(*C. Wesselhoeft*). Lithemic subjects; acid dyspepsia; cough ending with a loud belch.

Sulphur.—This remedy presents many of the symptoms which are likely to be encountered in a case of chronic bronchitis. It is especially applicable to the cough associated with an unresolved pneumonia. It has not proven of much use where emphysema is present, but where there is a large amount of tenacious mucus, mixed with lumps of pus, of foul taste and odor, it seems particularly applicable. There may also be attacks of oppression of breathing, in which the patient gasps for air.

Tart. emetic.—Useful in recent cases, with loud rales in the larger tubes, and dyspnea with the cough.

The *calcereas* are especially called for upon their diathetic indications.

Calc. carb., besides its characteristic sweat, large belly and glandular enlargements, will be indicated by loose cough, with expectoration of yellowish, sweetish mucus, or dry, teasing cough, with dyspnea and palpitation of the heart from slightest exertion. *Calc. phos.* is more suited to the purely rachitic case with diarrhea, or cases of simple malnutrition.

Silicea.—Emaciated children, tuberculous diathesis; night-sweats; profuse purulent expectoration; skin dry and scurfy; hectic fever; bronchiectasis; lack of normal body-heat, with constant chilliness. The cough is aggravated from cold drinks, and is deep and distressing.

Besides these it may be necessary to resort to one of the following remedies for special conditions and symptoms:

Ars.—Emphysema; dyspnea.

Carbo veg.—Hoarseness; chronic spasmodic cough remaining after whooping-cough. General loss of vascular tone of the entire mucous membrane of the respiratory tract.

Iodium.—Especially indicated in dark-complexioned, emaciated children. Ravenous appetite without a corresponding gain in weight; enlarged bronchial glands. The *iodides*, especially the *iodide of arsenic*, are useful in the bronchitis and accompanying wasting.

Kali bichromicum.—Tough, stringy expectoration; cough excited by eating. Bronchitis after measles.

BRONCHIAL ASTHMA.

True bronchial asthma is a manifestation of anaphylaxis to a foreign protein. The child has become sensitized to some protein, usually one of the food proteins such as casein, egg albumin, wheat, oats, potato, etc. The absorption of these proteins from the alimentary tract causes irritation of the constrictor fibres of the vagus which results in spasm of the circular muscles of the small bronchi. The asthmatic attack with its accompanying dyspnea and cough is thus explained. A foreign protein may also act peripherally by coming in

contact with the mucous membrane of the upper respiratory tract as in the cases of asthma accompanying hay fever or following exposure to horses, cats, flour, etc.

Asthma in children is usually associated with colds or bronchitis, even when the condition is one of anaphylaxis. The mucous membrane in childhood is more sensitive than in adults and reacts to a foreign protein very much the same as to a bacterial infection.

Walker (*Medical Clinics of N. Amer.*, Jan., 1918) has found that sensitization to animal and bacterial proteins occurs in the following order of frequency: horse dandruff, staphylococcus aureus, wheat and other cereals, the pollens, cat hair, egg, milk. This includes cases of all ages. Cases beginning in early life usually show signs of sensitiveness to some food protein; those occurring later may be due to bacterial sensitization or some non-food protein. Asthma developing after the age of forty is usually due to chronic bronchitis or cardiorenal disease.

Since the offending toxin may be of bacterial origin it is important to search for evidences of focal infection in the nose and throat and in the teeth in all cases. In Walker's experience the streptococcus hemolyticus is a frequent cause of the type associated with active bronchitis which he designates "asthmatic bronchitis."

Symptoms.—The attacks occur suddenly, usually at night, the chief symptom being dyspnea, accompanied by a dry cough and characteristic respiration. The inspiration is difficult, accompanied by recession of the soft parts of the thorax, and expiration is prolonged. The respiratory murmur is diminished, and the chest abounds in sibilant and sonorous rales; wheezing may be heard at quite a distance from the patient. Cyanosis becomes pronounced if the attack is a prolonged one. The attacks may last from a few minutes to an hour or more, and generally cease suddenly with a free secretion from the bronchial tubes; they recur at intervals of days or weeks.

The *catarrhal form* or asthmatic bronchitis is the type in

which asthmatic manifestations gradually develop during attacks of bronchitis which are of recurring character. Whether these cases are bacterial in origin, due to protein sensitization or to some disturbance of metabolism, is a difficult problem to solve. There is no doubt that a constitutional predisposition to asthma, often hereditary, exists and asthmatic children usually show evidences of disturbed metabolism and a neurotic constitution. A marked eosinophilia is present in those cases which are distinctly anaphylactic in origin. In some cases this is lacking and abnormalities in the nose and throat will usually be found in this type of asthma.

The **diagnosis** is readily made from the characteristic recurring paroxysmal attacks of dyspnea with prolonged wheezing expirations. Simple bronchitis, laryngismus stridulus and enlarged thymus must be excluded. The identification of the offending protein by means of the cutaneous test is often possible although the results are not as satisfactory in children as in adults.

Treatment.—The same hygienic measures recommended for bronchitis are applicable to overcome the tendency to recurring attacks of asthma. All foci of local irritation in the nasopharynx or elsewhere should receive prompt attention.

Every effort should be made to determine which particular kind of protein may be responsible and when found the same should be strictly eliminated from the diet. The gradual re-introduction of such a protein in small amounts into the child's diet will usually immunize the same against this food.

In some cases it is necessary to burn *stramonium* leaves in order to make the suffering endurable. In severe cases a hypodermic injection of adrenalin chlorid may be necessary.

Acon. is recognized by its well-known mental condition, feverishness, etc.; neurotic cases.

Apis.—When the attacks seemingly follow the recession of an urticaria, or alternate with the same. The chest feels bruised, and the attack ends with the expectoration of a large amount of frothy mucus and serum.

Ars.—Paroxysms between midnight and daybreak; must get out of bed; great anguish and prostration; bronchopneumonia.

Ars. iod.—Between the attacks. It is a valuable remedy in cases of asthmatic bronchitis.

Ipecac.—Wheezing; constant cough, with subcrepitant rales all over chest; no expectoration, although the chest seems full. Gagging and vomiting; the child stiffens during the choking attacks; cyanosis and coldness of extremities.

Lobelia.—In connection with disordered stomach; weakness in pit of stomach; attack preceded by prickling sensation in extremities; distressing tightness across upper portion of chest.

Nux vom.—Asthma dyspepticum; attacks in morning; irritability and constipation.

Pulsatilla.—Cough, becoming dry toward night, with dyspnea; inability to lie down; chilliness; mild, tearful disposition.

An autogenous vaccine, made from the bronchial secretion of the patient, especially if the staphylococcus aureus or a streptococcus can be isolated and grown, offers the best prospects of curing cases which are associated with distinct evidences of bronchitis, either chronic or of the recurring type.

ACUTE BRONCHOPNEUMONIA.

Bronchopneumonia, also known as catarrhal and lobular pneumonia, is one of the common diseases of childhood, presenting a mortality rate exceeded only by diarrheal diseases, and prevailing particularly before the fourth year of life.

Etiology.—There are two forms of bronchopneumonia, namely, primary and secondary. Primary bronchopneumonia occurs especially in infants debilitated by previous illnesses, or in those suffering from rickets or marasmus. Atmospheric changes are the chief exciting cause, as the greater prevalence of this disease during the winter and early spring months clearly indicates. Primary bronchopneumonia is less frequently seen after the fourth year, being essentially a disease of early child-

hood. It may, however, develop from the extension of a severe bronchitis at any age.

Secondary bronchopneumonia is a frequent complication of the acute infectious fevers, especially of the exanthemata, diphtheria, whooping-cough and influenza.

Bacteriological research indicates that primary bronchopneumonia is nearly always due to the pneumococcus, while secondary bronchopneumonia results from a mixed infection, in which the streptococci play the most important role. When complicating diphtheria the Klebs-Löffler bacillus is the excitant of the pathological process; the influenza bacillus may also cause bronchopneumonia.

Pathology.—In the larger bronchi a superficial inflammation is encountered while in the smaller tubes the entire wall shares in the pathological process, and we find here both bronchitis and peribronchitis. The characteristic lesions are in the air vesicles, which in typical cases are distended with cellular exudate. The cells are mainly swollen, desquamated epithelia with small nuclei. Red blood corpuscles and leucocytes are also found in variable number. Fibrin, as a rule, is scant; often entirely absent. The fibrin in these cases is difficult to demonstrate, as the threads are rendered indistinct through the presence of a large number of leucocytes (Ziegler). In the aveolar septa and peribronchial connective tissue the blood vessels are distended with red blood corpuscles and these structures are infiltrated with large mononuclear leucocytes.

Taking into consideration the above histological changes in the pulmonary tissue it is self evident that resolution must be often delayed, leading to permanent tissue changes. The co-existing bronchitis in the finer tubes explains the suffocative symptoms which may arise in capillary bronchitis.

Mixed types of pneumonia occur, in which one portion of the lungs is the seat of typical catarrhal and interstitial inflammation while another portion is consolidated by purely croupous exudation. These cases pursue more closely the clinical course of

bronchopneumonia than lobar pneumonia, but it requires microscopic examination to recognize the true character of the lesions.

Small bronchopneumonic areas representing consolidated alveoli may spread and become confluent, thus invading an entire lobule and giving rise to a lobular pneumonic process. These lobular areas are in the majority of cases separated by streaks of uninvaded lung tissue, i.e., lobules still pervious to air. An entire lobe may, however, become invaded, in which case we are confronted with a bronchopneumonia of lobar distribution (Ziegler).

The exudate in some instances is hemorrhagic in character. When resolution is delayed it frequently becomes purulent owing to the presence of a large number of leucocytes that have undergone degeneration.

Bronchopneumonia may abort in the early stage before consolidation can be detected and thus run the course of a severe bronchitis, or it may assume the clinical characteristics of a lobar pneumonia. Again, instead of undergoing resolution the inflammatory process may progress and interstitial pneumonia be the result.

The pathological findings are by no means uniform and as Delafield has pointed out the consolidated lobules may bear no definite relationship to the bronchus leading to them. The inflammation is diffuse in character, and lobule after lobule may become consolidated without its communicating bronchus being simultaneously involved. The inflammation therefore travels through contiguity of structure as well as by continuity thereof.

In the early stage (red pneumonia) the lung is engorged and of an intense red color. On section, a bloody, frothy fluid exudes from the air cells. The bases are heavier and darker in color owing to hypostatic congestion. Consolidation has not yet taken place, but microscopic examination reveals cell-proliferation in the peri-bronchial connective tissue and septa

and catarrhal and hemorrhagic exudate in the alveoli. The process may abort here, prove fatal, or go into the stage of mottled or red and gray pneumonia, representing the fully developed process. If the lung is examined at this stage, both the surface and the sections will present a mottled appearance due to the admixture of consolidated (gray) and congested (red) areas. The process may involve an entire lobe or appear only in patches dispersed through the otherwise normal lung tissue. Wherever a bronchus has become occluded areas of atelectasis are seen.

If resolution be delayed or arrested, the so-called gray pneumonia results. In these cases the lung is somewhat enlarged, gray in color and extensively consolidated. Pleural thickening and adhesions are common. On section a mucopurulent exudate covers the cut surface. The bronchial walls and the interstitial tissue are hyperplastic and areas of atelectasis and compensatory and interstitial emphysema lie interspersed between the consolidated areas.

In the cases that recover the termination is resolution through expectoration and resorption of the exudate; in unfavorable cases suppuration; interstitial induration; gangrene.

Resolution may begin before consolidation can be detected. Ordinarily it is completed in from two to three weeks. When delayed, there is a strong tendency to incompleteness of the process. In recurring attacks, permanent interstitial changes are apt to remain.

The pleura shares in the inflammatory process when the lesions are superficial. Fibrinous and fibro-purulent exudate is poured out upon the surface of the visceral pleura with the consequent development of adhesions and thickening. In some instances the pleuritic process becomes a prominent feature of the case (pleuro-pneumonia). This is more common, however, in the lobar type of the disease.

Symptoms.—Primary bronchopneumonia begins clinically as a bronchitis in the majority of cases. Instead of advancing

favorably, however, there develops progressively increasing dyspnea and rapid breathing, increase in fever and pulse-rate, and prostration.

Some cases begin, like lobar pneumonia, abruptly, with chill, high fever, rapid breathing and pronounced nervous disturbance (toxemia). They may prove fatal before signs of pulmonary inflammation have had time to develop; even cough may be absent. At the autopsy the lungs are found intensely congested and edematous.

In young infants bronchopneumonia may come on insidiously, fever being slight during the entire course. The main symptoms are cough, progressively increasing cyanosis and rapid respirations. As a rule, gastro-intestinal symptoms accompany the pneumonia. The prognosis is grave.

During the progress of the disease the child emaciates markedly and carbonization of the blood becomes apparent. The pulse is rapid and weak, and the heart may eventually fail in its work if the pulmonary obstruction is extensive.

Cough is a prominent symptom; in the beginning it is hard and dry; later it sounds loose but since young children are unable to expectorate the cough gives little relief. Much of the mucus, however, is swallowed.

Respiration is often accompanied by fan-like movements of the alæ nasi and recession of the soft parts of the thorax, notably its lower portion. In rachitic infants serious deformity of the chest results.

When bronchopneumonia develops during the course of one of the infectious fevers it is to be suspected from an increase in the fever, increased rapidity of breathing and pulse-rate, cough and dyspnea, especially the latter.

Bronchopneumonia tends to localize itself in certain areas of the lungs, in this way differing from simple bronchitis in which the process is general.

In the absence of signs of consolidation, the height and duration of the fever are the symptoms upon which we must

rely in diagnosing bronchopneumonia. Cabot states that bronchopneumonia should be suspected in the adult when the patient is too sick to have bronchitis, and this applies with equal force to children.

Bronchopneumonia is progressive in its development, being slower both in its onset and in the development and resolution of its pathologic products than lobar pneumonia. Its course sometimes extends over several weeks, and protracted cases are common, especially in the debilitated.

Meningeal symptoms, either of toxic origin or due to an associated meningitis, may occur, but are not as commonly encountered as in lobar pneumonia (see "Cerebral Pneumonia").

Death results from respiratory or cardiac failure; sometimes from hyperpyrexia. Collapse is the commonest termination; sometimes death occurs with convulsions or coma. The fulminating cases die from toxemia.

The *prognosis* must always be guarded, as can be seen from the high mortality rate. It is especially grave when the child is very young and debilitated, or when the disease is secondary to a condition in itself dangerous. The pulse and respiration are the main indications of the child's condition. A continuously high temperature is more dangerous than one in which remissions occur. Cyanosis is always a grave symptom. The soft condition of the chest-wall in rickets makes breathing very difficult in pneumonia and rachitic children stand the disease badly.

A grunting expiration is said to indicate atelectasis, but is not necessarily a bad symptom, unless very pronounced and persistent. It may be due to an associated pleurisy. The cough is also a guide to prognosis; when it becomes weak and ineffectual and mucus collects in the larger tubes it is usually a sign of oncoming respiratory failure.

Diagnosis.—The physical signs are those of bronchitis of the larger and smaller tubes associated with consolidation of scattered areas of pulmonary tissue of varying size and extent.

They are best elicited posteriorly, the child being held over the nurse's shoulder. One may detect large and small moist rales; subcrepitant rales; tubular breathing and dullness over the consolidated areas of sufficient extent to convey these signs (usually the bases of the lungs); diminished breathing over areas of atelectasis, and exaggerated breathing in the vicariously emphysematous lung. In the absence of distinct physical signs, however, the presence of fever, cough, rapid labored respirations and prostration are sufficient clinical evidence upon which to make a diagnosis of bronchopneumonia.

From *croupous pneumonia* it is distinguished by its gradual onset, tedious course, bilateral distribution, absence of extensive lobar consolidation, and its occurrence in the very young and in the feeble, croupous pneumonia usually attacking those in apparently good health and of more mature age. In *capillary bronchitis* there are fine moist rales generally distributed throughout the chest. There is, however, no sharp line of demarcation between the pathology of the two affections. *Tuberculosis* is more gradual in onset and when physical signs develop they are more prominent in the apices than at the bases. There is no tendency toward recovery as in bronchopneumonia and the dyspnea is out of proportion to the physical signs present. Other evidences of tuberculosis may be detected, such as the signs of enlarged bronchial glands, and the sputum may show the presence of tubercle bacilli.

Treatment.—The child should be put to bed, and its position changed regularly to avoid adding hypostatic congestion to the already seriously crippled condition of the lungs. Infants can be taken up by the nurse during coughing paroxysms and held face downward or on the side to facilitate the expulsion of the bronchial secretion. The room must be thoroughly ventilated, and a temperature of about 60 to 65 degrees maintained. The "cold air" treatment is contraindicated in cases with much bronchitis but it is of decided value in the croupous type of pneumonia. In cases of "capillary bronchitis" it is important

to moisten the air in the immediate vicinity of the child. This is best accomplished by means of the croup-kettle or steam spray. High fever is best combated by fresh air, cool or tepid sponge baths and bowel irrigations. In hyperpyrexia a cold pack may be used. When carbonization of the blood becomes manifest and the bronchial tubes become clogged with secretion, the alternate application of hot and cold packs to the chest should be resorted to. A warm full bath, followed by a cool sponge bath, is frequently useful to relieve the pulmonary congestion.

Oxygen inhalations may be employed in serious cases. From one to two gallons (bagfuls) administered by holding a glass funnel attached to the tube from the water bottle of the apparatus over the child's mouth and nose may be given every hour. There is no objection to enveloping the chest in a cotton jacket if the sick room is to be kept cool and if there is an associated pleurisy. When, however, the child is kept in a warm, moist atmosphere on account of the bronchitis and cough, care should be exercised not to have it overdressed.

In case of collapse, brandy or aromatic spirits of ammonia may be resorted to. Camphorated oil, five minims, hot packs to the chest, and oxygen should be used if the collapse and cyanosis persist. It may become necessary to use brandy or whiskey regularly in small doses in protracted cases with failing circulation.

The remedies most frequently indicated in the early stages are *acon.*, *bell.*, *bry.*, *ferrum phos.*, *ipsecac* and *scilla*; for the later manifestations, especially the unfavorable symptoms likely to arise, *tartar emetic*, *phos.*, *arsen.*, *carba veg.* and *veratrum alb.* are called for.

Aconite should always be studied in comparison with *veratrum viride* and *ferrum phos.* All three are indicated early in the disease, when there is high fever and a teasing cough, with little or no expectoration—the stage of congestion. *Aconite* is distinguished by its great anxiety and restlessness, thirst, and aversion to being touched or moved, which induces suffering;

veratrum viride by its vascular excitement and respiratory embarrassment, bloodshot eyes and cerebral irritation; *ferrum phos.* by the absence of either nervous erethism or high arterial tension and by its characteristic frothy, blood-streaked expectoration.

Arsenicum is indicated by extreme prostration and restlessness; dyspnea from the slightest exertion; thirst for small quantities of water, the mouth being dry and the tongue and lips cracked; diarrhea; cold surface.

Belladonna is particularly valuable when nervous disturbances are pronounced. Its excellent effect in capillary bronchitis makes us think of it in pneumonia when the bronchial symptoms predominate. *Belladonna* is homeopathic to the vascular engorgement and high temperature so prominent in many cases.

Bryonia is of the greatest service to loosen the cough, control the pain, and check the extension of the process into the smaller tubes and promote the absorption of the exudation. It must be differentiated from *scilla*, which is similar in many respects, but more suitable to grave cases marked by progressively-increasing prostration and dyspnea; rapid, weak pulse; short, painful cough, causing the child to cry faintly after each paroxysm; in fact, it cannot be moved without giving it pain. Hale (*Practice of Medicine*) considers *scilla* the remedy above all others after *aconite* and *belladonna*, being in every respect homeopathic to bronchopneumonia.

Ipecac is the remedy where the bronchial element predominates and the chest seems literally filled with mucous secretion, subcrepitant rales being heard everywhere in abundance. The cough is troublesome and gagging, giving little relief. The secretion gradually collects to such an extent in the finer bronchi that suffocation becomes imminent. In this regard it differs from *tartar emetic*, which represents a state of carbonic acid poisoning, in which mucus, collecting in the larger tubes, produces the characteristic rattling, or in which there is active pulmonary edema.

Phosphorus.—Where consolidation predominates over the bronchial symptoms, together with active congestion, producing a tight, distressing cough; rapid, shallow respirations; tightness across the upper portion of the chest; blood tinged expectoration; failing right heart. We are inclined to think of *phosphorus* only in lobar pneumonia, but it is of equal value in the lobular variety when we have to deal with congestion, consolidation and toxemia; in fact, some old school writers (Wood) recommend *phosphorus* as a nerve tonic in the adynamia of pneumonia.

Sulphur is similar to *phosphorus* in its effect on consolidation, but it has a greater power of removing the same. *Phosphorus* mainly controls the vascular disturbance. *Sulphur* is indicated in the later stages of bronchopneumonia.

CROUPOUS PNEUMONIA.

Croupous, or lobar pneumonia, is a primary acute infectious disease in which one or more of the pulmonary lobes are consolidated by a croupous exudate. It is a self-limiting disease and is not accompanied by bronchitis.

Etiology.—Croupous pneumonia is most frequently seen after the third year, and usually attacks those of previously good health, unlike bronchopneumonia, which attacks with predilection those already debilitated or develops in connection with the acute infectious diseases. I have, however, encountered it in infants as young as two and three months old. Fatigue and exposure to cold act prominently as predisposing causes. While the dry, cold months, particularly the early spring, furnish the largest number of victims, still pneumonia may be seen at any time of the year, like all other infectious diseases. Boys are more often attacked than girls.

Recently, studies of the organisms present in pneumonia in children have been made with the purpose of determining the type of pneumococcus responsible for the different cases. Pisek and Pease (*Amer. Jour. Med. Sciences*, 1916), found type 1 nine times, type 2 eleven times, type 3 once, type 4 seven times

in twenty-eight cases studied. Wollstein and Benson (*Amer. Jour. Dis. Child.*, 1916), found type 4 in 60 per cent of the cases with a mortality of 40 per cent. This is quite different from the findings in adults in whom type 4 is not often fatal.

Pathology.—In typical cases of croupous pneumonia one lobe is affected in its entirety. The most frequently consolidated lobe is the left lower; next in frequency come the right lower and the right upper lobes. The right middle and the left upper are the least often attacked.

Some degree of plastic pleurisy is always associated. When the lower left lobe is affected and the pleura is involved the process may spread to the pericardium. The pleural inflammation may become so prominent as to influence notably the clinical course of the disease.

At the onset of pneumonia, the stage of engorgement, the affected lobe is bright red, greatly congested and somewhat edematous. The lung appears enlarged, as if inflated, and when the inflammatory exudate fills the alveoli and solidifies, the consolidated lobe is actually larger than normal, for which reason the area of dulness elicited by percussion may be of greater extent than that which the lobe normally occupies.

On microscopic examination the alveoli appear engorged, the bloodvessels encroaching upon their lumen. A small amount of serum and leucocytes has been poured out and the exudation becomes more and more rich in cells and fibrin and more hemorrhagic in character as the process goes on. At this time the crepitant rale is most clearly heard. The alveoli eventually are distended to their utmost with red and white blood corpuscles embedded in a stroma of fibrin. The fibrin also fills the lymphatics in the interstitial connective tissue, and it can be seen communicating by thin bands through the pores of the alveoli. This period represents the stage of red hepatization.

The color of the lung gradually changes from red to gray, owing to the compression of the bloodvessels of the alveoli by the exudate and to the degeneration of the cellular elements.

This stage is called gray hepatization. Later the exudate is gradually removed by the lymphatics, some being expectorated after having undergone softening, and resolution is thus established. In the usual cases resolution becomes complete and the lung is restored to its normal condition.

During consolidation the lung is quite friable and cuts like liver. On the surface of the section small plugs of hardened fibrin filling the alveoli and independent therefrom are seen, giving it a granular appearance. In children this does not show as typically as in adults, owing to the lesser development of the air cells. At times, owing to a gradual spread of the process, all stages, that is, red and gray hepatization and beginning resolution, may be encountered in a section of a single lobe.

When resolution is delayed it may terminate in suppuration with abscess formation, gangrene, caseation. Complete recovery is, however, the rule, excepting in cases complicated with pleural inflammation, in which it is common for an empyema to develop secondarily.

Symptoms.—The onset of croupous pneumonia is characteristically sudden, and the course of the disease is acute throughout; sudden onset, high temperature, with but slight remissions and termination within from six to eight days by crisis are the features of a typical case. In the majority of cases the crisis occurs on the seventh day.

The initial symptom is a chill, which may be replaced by a convulsion in young children. Sometimes vomiting is the sole symptom. In young children the chilly stage becomes manifest by a cyanotic pinched appearance and noticeably cold hands and feet. The temperature rises rapidly, soon reaching a height of 104 degrees or higher; the pulse is rapid and full, and the respirations are strikingly increased, exceeding the normal ratio between pulse and respiration. Thus, with a pulse of 120 there will be 60 or more respirations, while the normal ratio is one respiration to four heartbeats. The temperature range may vary between 102.5° to 105°. Remissions are more pronounced than in adults.

Associated with the fever there is restlessness; dry, hot skin; headache and some delirium toward night, and a dry, painful cough. The face wears a characteristic distressed expression. When there is considerable involvement of the pleura the painful cough becomes a prominent feature of the case. When the parietal pleura of the chest wall is inflamed, there results a sharp stabbing pain at the site of the inflammation which is made worse by coughing, deep breathing or even muscular movements affecting the chest wall. Capps has shown that when the diaphragmatic pleura is inflamed at the peripheral portion of the diaphragm, the pain is referred to the lower portion of the thorax and to the abdomen, as the result of irritation of filaments of the lower intercostal nerves. This has frequently led to the erroneous diagnosis of an abdominal affection, notably of appendicitis, when the right lower lobe is affected. With involvement of the central portion of the diaphragm the pain is referred to the neck and may cause symptoms likely to be confused with meningitis (rigidity of the neck).

Within from two to four days the process of consolidation is generally complete, as can be demonstrated by the dulness and bronchial breathing observed over the affected area. With the crisis, which may appear on any day from the fifth to the ninth, oftenest, however, on the seventh day, there is a marked amelioration of all symptoms. A profuse sweat accompanies this sudden fall in temperature, and at times, indeed, there occur quite alarming symptoms of collapse, calling for immediate action. After the crisis the process of resolution becomes established, being completed in from five days to a week in the average case. A rise of temperature during this time—in other words, a post-critical rise—indicates the development of some complication, such as pleurisy, empyema, meningitis, pericarditis or the extension of the pneumonic process to other portions of the lungs. A pseudo-crisis is common in children; it usually occurs on the fifth day. Termination by lysis is more frequent in children than in adults, but is atypical. A pro-

tracted course means a complication, usually an associated pleurisy. Marked remissions in the temperature are also more common in children than in adults. When pronounced these cases are described as remittent pneumonia.

The blood changes are important. While there is but a slight anemia, leucocytosis develops to a marked degree. A pronounced leucocytosis indicates a severe infection in an organism capable of good reaction. There is usually a rise in the leucocytes to 20,000 and over. Leucocytosis offers a reliable sign of differential diagnosis between pneumonia and such conditions as acute typhoid septicemia, influenza, caseous pulmonary tuberculosis and serous pleurisy, it being absent in these conditions. It is of no value, however, in the differentiation of croupous pneumonia from bronchopneumonia, empyema and cerebrospinal meningitis.

Many severe cases of pneumonia present a clinical picture so different from the group of symptoms above enumerated that they merit separate description, being classified into the following varieties:

Cerebral pneumonia.—This form is characterized by rapid onset with fever, convulsions or vomiting, and a predominance of cerebral symptoms during the entire course of the disease. In children over three years old convulsions may be absent, but delirium and coma develop. Symptoms simulating meningitis, such as stupor, strabismus, opisthotonus, slow irregular pulse, dilated pupils, and convulsions, are a frequent accompaniment of the severe types of pneumonia, and there seems to be a close clinical relationship between pneumonia of the upper lobes and cerebral symptoms. The pneumonic process is slow to develop in many cases, and often the consolidation cannot be detected until the fourth or fifth day, for which reason meningitis is suspected. The brain symptoms rapidly subside after the crisis. In cases of meningitis complicating pneumonia the brain symptoms develop during the course of the pneumonia and are not present at the time of onset, and are uninfluenced by the crisis.

Wandering pneumonia.—Another form of pneumonia worthy of mention is the so-called *wandering pneumonia*, in which the pneumonic process spreads from its original seat to other portions of the lung, resolution going on at one point while a fresh invasion occurs in another.

Central pneumonia is of special interest from the diagnostic standpoint, since it has long been held that cases of pneumonia in which physical signs are absent or late in developing are of this type and that the pathological process begins in the central portion of the lung and gradually extends to the surface. This, however, is a pathological fallacy and the X-ray has demonstrated that the shadow cast by a pneumonic consolidation is triangular in shape with the base in the axilla and the apex toward the hilum. As soon as the consolidation extends far enough down into the parenchyma of the lung to come in contact with a large bronchus, bronchial breathing will be heard, but prior to that time there will be no distinct evidence of pulmonary consolidation (*Mason, Amer. Jour. Dis. Child., 1916. 11*). It is clear that these cases cannot be diagnosed until there is sufficient basis for their recognition. Grave symptoms may exist with only a small amount of consolidation, the toxemia being entirely out of proportion to the existing lesion.

Pneumonia with Gastrointestinal Symptoms.—One of the characteristic features of pneumonia in young children is the prominence of gastrointestinal symptoms. These may completely mask the clinical picture, so that the pneumonia is not suspected. Vomiting, diarrhea, distended abdomen and high fever not responding to appropriate treatment directed to the alimentary tract should arouse a strong suspicion of pneumonia and lead to a careful examination of the chest. The grunting respiration of the pneumonia may be mistaken for the child's ineffectual urging to stool, or be interpreted as an embarrassment of respiration resulting from the tympanites, thus adding to the confusion.

Influenzal pneumonia may be due either to the Pfeiffer bacillus or result from secondary infection with the pneumococcus or streptococcus during an attack of grippe. These cases begin as an influenzal bronchitis, during the course of which one or more pulmonary lobes become consolidated. The course is graver and more protracted than in primary lobar pneumonia.

Abortive pneumonia is rare in children. Cases are encountered which terminate in from four to five days; they might be called mild cases. Again, the process may not go beyond the first stage, and although congestion of a single lobe and pneumococci in the sputum can be demonstrated, consolidation fails to take place, the process actually aborting, as other acute infections sometimes do. It is needless to say that the diagnosis is beset with great difficulty. There are also *fulminating* cases, terminating fatally in the first twenty-four hours.

Typhoid-pneumonia.—This misleading term refers to that type of pneumonia in which the patient sinks into a typhoid state as the result of the toxemia. Instead of active brain symptoms being present as in cerebral pneumonia there is apathy and prostration; dry, coated tongue; tympanites; involuntary stools; muttering delirium; subsultus tendinum. Rose-spots, enlarged spleen and Widal reaction are absent. Typhoid fever, however, may be complicated with pneumonia. In doubtful cases blood cultures should be made.

Pleuro-pneumonia is a form of pneumonia (6.8 per cent in Holt's series of 398) in which pleurisy coexists with the pneumonic process and to such an extent as to affect the clinical course of the disease in a decided manner. The pleural inflammation is chiefly plastic in nature and the growth of serum poured out is relatively slight; never so great as seen in a primary pleurisy. At the autopsy the pleural surfaces are found matted together and covered with a thick, yellow, plastic exudate that can be readily scraped off and from the interstices of which turbid serum exudes.

The surface of the entire lung on one side may be covered

with this exudate even though only one lobe be consolidated. The changes in the lung are not necessarily lobar, but may be bronchopneumonic in type. Cases which recover develop a subsequent empyema. The mortality rate is very high.

In the first stage there is every evidence of a beginning pneumonia, together with severe pain in the side and the physical signs of pleurisy. Friction sounds are plainly heard and in the course of a few days distinct dullness, bronchial breathing and bronchophony can be detected. These latter signs are somewhat obscured by the thick fibrinous layer, but never to the extent produced by an effusion. Aspiration is negative, but the symptoms are too severe for a simple pleurisy and too indistinct for a frank pneumonia. The prognosis is unfavorable, especially in young children. Pericarditis is a common complication. Cases that survive may have to go through the course of an empyema with possibly severe crippling of the lung. When, however, the process remains localized, perfect recovery, barring adhesions, is possible.

Complications.—A certain amount of pleurisy accompanies all cases of pneumonia. Pleural effusions, both serous and purulent, are, more strictly speaking, sequelæ; they are much more common in children than adults. Otitis is rare; it is, however, frequently seen as a complication of bronchopneumonia. Meningitis is more likely to occur late in the case. Cerebral symptoms coming with the onset or early in the case, are more likely to be of toxic origin. Pericarditis is a grave complication; it is seldom recognized in *vitam*. Other complications that may develop are myocarditis, endocarditis, peritonitis, gastro-enteritis, arthritis, septicopyemia.

Physical Signs.—The physical signs in lobar pneumonia vary with the different stages of the pathological process. The duration, clinical course and complications also modify these signs as well as the age of the child.

In the *first stage* the child appears flushed with fever and the respirations are quickened. There is characteristically a

short inspiration followed by a pause and then a quick panting or moaning expiration. The pulse-respiratory ratio are changed from the normal 1 to 4 to 1 to 2.

Over the affected lobe the respiratory sounds are feeble, sometimes almost suppressed, while percussion reveals a slight dulness of tympanitic quality. On the opposite or well side the breath sounds may be harsh and exaggerated, often leading to the error of suspecting the lesion on the well side. On coughing the crepitant rale may be heard.

Friction rales, pleural in origin, are frequently heard. An interesting observation has been made by Shaw (*Archives of Pediatrics Aug., 1903*), who found that the crepitant rale and friction can be distinctly heard over the abdomen when the lower lobes are affected.

Second Stage.—With the completion of consolidation vocal fremitus is increased over the affected lobe and percussion dulness becomes pronounced. The area of dulness apparently covers a larger area than the anatomical boundaries of the lobe allow for. This is explained by the fact that the croupous process distends and enlarges the lobe. When pleural effusion takes place the lower portion of the dull area becomes flat. It is not uncommon to hear friction sounds in the lower part of the chest, posteriorly and laterally, in pneumonia in this region. The adjoining normal lung, through compensatory emphysema, may give the vesiculo-tympanitic note. Auscultation reveals bronchial breathing and bronchophony.

Third Stage.—As resolution sets in and the exudation begins to soften, crepitation reappears (*crepitatio redux*). Moist rales are usually added and considerable of the exudate is coughed up. Bronchial breathing persists longer than actual consolidation; so also dulness. This is no doubt due to the relaxed and congested state of the pulmonary tissue. For this reason it is possible to demonstrate abnormal physical signs for some days after the crisis. We must, however, regard with suspicion the persistence of pronounced dulness and diminished or absent

respiratory murmur after pneumonia. Delayed resolution should always suggest the probability of fluid being present.

Prognosis.—In infants the prognosis is less favorable than in older children. Robust children from three to ten years old recover as a rule. Indeed, the mortality rate at this period of life is strikingly lower than in adults. The type of organism responsible for the infection bears a strong relationship to the prognosis.

Of primary importance in gauging the prognosis is the degree of toxemia. This seems more important than the extent of the pulmonary involvement or the height of the fever. Naturally, the spread of the disease to adjacent portions of the lung is unfavorable. The heart holds out better than in the adult because the child's circulatory apparatus can adapt itself to increased circulatory obstruction better than the adult's.

The majority of deaths occur at the height of the disease. When death occurs later it is the result of one of the above mentioned complications.

Diagnosis.—Whenever we are confronted with an acute condition of sudden onset with high fever preceded either by a chill, vomiting or a convulsion, we should first examine the throat. If this is found to be negative the chest should be carefully investigated. Many cases of high fever of sudden onset clear up in a day or two, and cannot be accounted for. Sometimes an acute otitis causes symptoms simulating the onset of pneumonia, even convulsions being present, all, however, clearing up after the ear begins to discharge. We should always suspect the lungs in the presence of continued high fever and increased respirations, especially if the expiration is of the characteristic moaning type.

The conditions from which pneumonia is to be differentiated are bronchopneumonia, pleurisy, meningitis and caseous tuberculosis. *Bronchopneumonia* is essentially bronchial in origin, both etiologically and pathologically, and its course is long and protracted, independent of complications. In *pleurisy* the

physical signs are essentially different and the onset gradual. The fever is not so high and terminates by lysis. Besides, primary pleurisy with effusion is rare in children, but pleuritic inflammation with purulent exudate secondary to pneumonia is common.

In *meningitis* symptoms are continuous and protracted. Death is practically always the termination excepting in the epidemic cerebro-spinal variety. Meningitis complicating pneumonia occurs in the later stages of the disease; cerebral symptoms occurring at the height of pneumonia are toxic and disappear by crisis or even before the crisis.

Typhoid fever beginning abruptly may cause confusion. The absence of leucocytosis and the later appearance of rose spots, the Widal reaction and enlarged spleen positively identifies it.

Acute caseous pulmonary tuberculosis may set in with a chill and lead to the consolidation of an entire pulmonary lobe within a remarkably short time. The temperature will run high and the entire clinical picture be identical with that of croupous pneumonia. Crisis does not occur, however, and eventually softening and break down of pulmonary tissue sets in. Elastic fibres and tubercle bacilli can be detected in the sputum at this time, confirming the diagnosis. The most experienced are deceived, however, in the early stage of such a case.

Treatment.—The treatment of croupous pneumonia is essentially the same as that recommended for bronchopneumonia. The fresh air treatment may be carried out without restriction, as there is no contraindication to cold air like in some cases of bronchopneumonia. There are certain remedies which are especially related to croupous exudations, in contradistinction to those of a purely catarrhal type, and they will, therefore, be more useful in croupous than bronchopneumonia. Thus, *ipecac* and *tartar emetic* are less frequently indicated than *bryonia* and *sulphur*. In the early stages *aconite* is the most useful drug. When blood streaked sputum is present *ferrum phos.* may be used as a routine remedy.

Iodine is recommended by Kafka (*Homeopatische Therapie*) as being truly homeopathic to the croupous exudation, as well as to most of the symptoms.

The high fever and cerebral symptoms will call for *bella-donna* or *veratrum viride*.

Bryonia is especially valuable in pleuropneumonia.

Although *phosphorus* is more useful in bronchopneumonia than in croupous pneumonia, still it is of good service where there is marked congestion indicated by dyspnea; tightness across the upper portion of the chest; bloody expectoration; failing right heart and profound toxemia.

Sulphur is the chief remedy to aid resolution, being especially useful in the third stage of pneumonia.

Arsenicum is well suited to those atypical cases in which the poison of influenza is added to that of pneumonia. In the presence of abundant bronchial secretion with dyspnoea and cardiac weakness, the *iodide of arsenic* is preferable.

Special symptoms are to be dealt with as directed under *bronchopneumonia*.

PULMONARY TUBERCULOSIS.

Tuberculosis of the lungs in childhood is encountered in a variety of forms which may be either acute or chronic in their course. During infancy acute miliary tuberculosis of the lungs and acute tuberculous bronchopneumonia are the types usually seen. Older children show more resistance toward the tubercle bacillus and as a rule the infection remains latent at this time of life. When this is the case the clinical manifestations are chiefly those of involvement of the bronchial glands. A tuberculous bronchopneumonia, however, of less acute course than in infancy may be encountered at this age. During late childhood the open form of chronic pulmonary tuberculosis, or phthisis is seen as in the case of adults but it does not become a common affection until the period of adolescence is reached.

Acute Miliary Tuberculosis.—During infancy a dissem-

inated miliary tuberculosis is a frequent terminal condition developing secondarily from a primary focus of infection in the lung. The bronchial glands are incapable of arresting the infection as in the case of older children and for this reason miliary tuberculosis readily develops in an infant. Miliary tuberculosis of the lungs may also be a terminal event in a case of chronic pulmonary tuberculosis in an older child.

Tuberculous bronchopneumonia may be encountered at any period of childhood and it may occur primarily from the extension of the infection in a bronchial gland or it may develop secondarily after an attack of bronchopneumonia or an acute infectious disease. From the standpoint of the pathologist bronchopneumonia always develops secondarily to a primary focus of infection in the lungs and bronchial glands. Such apparent exciting causes as an attack of pneumonia or measles, for example, act through lowering the child's resistance and in aiding the breakdown and extension of the primary lesion. There are two types of tuberculous pneumonia, namely the *bronchopneumonic type* and *acute caseous tuberculous pneumonia*. In the former, tuberculous nodules and caseous areas of varying size are formed throughout the lungs, one lung, however, usually being more involved than the other. These areas of consolidation more frequently occur in the upper lobes than in non-tuberculous bronchopneumonia. In some instances a lower lobe may rapidly become caseous throughout, thus simulating lobar pneumonia. Instead of resolution, however, setting in as in the case of the latter disease the lung eventually breaks down and cavities may form in the consolidated area. The consolidation results from epithelial infiltration of the alveoli (desquamative pneumonia). Bronchitis and peribronchitis are associated processes. The solidified areas undergo caseation and these in turn break down as a result of necrosis or secondary infection with pyogenic organisms.

Symptoms.—Tuberculous bronchopneumonia may develop primarily by extension from infected bronchial glands or it may

occur as a sequel to some acute infectious disease or after an unresolved pneumonia. It presents the symptoms of an ordinary bronchopneumonia in its early stage, there being cough, continued fever and the physical signs of bronchitis and areas of pulmonary consolidation. The apices are more frequently involved than the bases of the lungs and the sign of d'Espine can usually be demonstrated. At first dulness and persistent subcrepitant rales in the consolidated areas are elicited; later the rales become resonating in character and bronchial breathing develops.

The temperature is more remitting than in ordinary bronchopneumonia and the course of the disease is more protracted. In infants, however, it usually runs an acute course. The fever is not as high as in bronchopneumonia and there is less toxemia. Cyanosis develops in the latter stages of the disease.

The child emaciates rapidly and anemia is pronounced. The cough is persistent and may be paroxysmal in character owing to the enlargement of the bronchial glands. Expectoration and hemoptysis are usually absent. The duration is variable and may be protracted to two or three months with apparent remissions in the disease.

Diagnosis.—Bronchopneumonia running a protracted course with predominance of physical signs in the upper lobes and interscapular dulness should suggest tuberculosis. This is especially true of pneumonia developing after measles or whooping cough. The same holds true of a lobar pneumonia which fails to undergo resolution. The probabilities are greater for the condition being primarily tubercular than of having become secondarily so. Dulness is more pronounced over a tubercular than over a pneumonic consolidation; the former, in fact, may suggest fluid and it is at times necessary to resort to the use of the aspiring needle to differentiate these conditions. A family history of tuberculosis and the tuberculous diathesis, or a history of exposure to a tuberculous source of infection, offer strong presumptive evidence.

Positive evidence is offered by finding the bacillus of Koch in the sputum. This diagnostic sign is, however, not always available, owing to the difficulty of obtaining sputum. A satisfactory method of obtaining the sputum for microscopic examination is suggested by Holt. A catheter is inserted several inches into the esophagus after a coughing spell, by means of which sufficient sputum can be obtained, as children invariably swallow their expectoration. This is a simple and perfectly reliable procedure and one that should never be neglected in suspicious cases. A piece of gauze, held in the jaws of an artery clip, also answers very satisfactorily for obtaining sputum from the pharynx.

Treatment.—The *treatment* is the same as for bronchopneumonia.

Remedies are unfortunately of little help. *Iodine* 3x dilution is the best indicated remedy, the symptoms calling for it being high fever, cough, rapid pulse and respirations and emaciation. Other remedies which may relieve some of the symptoms present are *arsenicum*, *phosphorus* and *sulphur*. The prognosis is always unfavorable although the disease may assume a chronic course or at times become apparently arrested.

CHRONIC PULMONARY TUBERCULOSIS.

The chronic form of pulmonary tuberculosis, in which fibrosis is added to the caseous process, is seldom encountered before the sixth year, not becoming a common disease until after puberty. Its course is identical with that of cases of consumption in young adults.

A variety of lesions is found, the characteristic pathological changes being caseation and fibrosis in conjunction with cavity formation. Owing to the tendency to destruction and excavation of pulmonary tissue, the terms "ulcerative phthisis," and "open tuberculosis" are frequently employed to designate this disease. The coexistence of miliary granulations and areas of caseation and fibrosis indicates that the course has been marked by

remissions, as well as periods during which the pathological process has been active. Such a period of activity often occurs immediately before the death of the patient, and during its continuance miliary tubercles in great number may form in parts of the lung hitherto unaffected (Fowler).

The seat of the earliest lesion is one of the apices, in the majority of cases the right. The process does not begin at the extreme apex of the lung, but about an inch below that point, and nearer the posterior and external than the anterior border, spreading thence backwards. The upper and posterior part of the lower lobe is involved often long before extensive infiltration or destruction of the upper lobe has taken place, and, as a rule before the apex of the opposite lung is attacked. Infiltration of the lung at this site, together with infiltration of the apex, is almost positive proof of the existence of tuberculous disease of the lungs (Fowler).

Associated lesions usually found are bronchitis, peri-bronchitis and bronchiectasis; emphysema (compensatory); pulmonary collapse, the result of bronchial obstruction; edema and congestion at the bases; pleurisy, usually of the chronic proliferating type. Lesions in other organs that may be encountered are tuberculous ulceration of the intestines, amyloid disease of the internal organs, tuberculous adenitis, meningitis and tuberculous arthritis.

Females seem more prone to consumption than males. The ages between twenty and thirty furnish the highest percentage of cases, the number gradually increasing from the fifth year to that time.

Certain previous diseases invite it. An attack of acute pleurisy often precedes the outbreak of pulmonary tuberculosis, or a lung impaired by a former pleurisy may become susceptible. Recurring attacks of bronchitis, an unresolved pneumonia, influenza, measles and whooping-cough are predisposing factors.

Symptoms.—The first indications of failing health to attract attention to the lungs may be a gradual *loss of weight*.

Hemoptysis is a very suggestive symptom but it is not as frequently seen in children as in adults. *Fever* is usually present but is often unsuspected. The temperature may be subnormal in the morning and rise to 100° to 101° in the evening (rectal). *Night sweats* occur in the later stages when secondary infection has set in. The *pulse* is soft and rapid even when there is no fever.

Physical examination reveals an emaciated frame; long, flat chest and superficial, feeble respiratory movements. The absence of the typical phthisical chest does not, however, exclude the possibility of pulmonary disease. When the process is active, the skin is dry and feverish. Commonly, enlarged superficial lymphatic glands can be felt in the neck and supra-clavicular region. The clavicles stand out prominently, as do also the scapulæ, and the infra-clavicular region is flattened. Palpation reveals increased vocal fremitus in either one or both infra-clavicular regions; the percussion note is dull in the supra-clavicular region, and the area of dullness often extends down as far as the third rib anteriorly, occupying the inter-scapular space on one or both sides of the spinal column posteriorly. The dullness may be associated with a suggestion of tympanitic quality. Auscultation reveals, in the early stages, harsh breathing in the affected apex, associated with fine, crackling rales. Broncho-vesicular breathing soon develops. As infiltration advances, bronchial breathing can be elicited in the infra-clavicular space. This can usually be demonstrated earliest posteriorly at a point opposite the fifth dorsal spine, midway between the border of the scapula and the spinous processes of the vertebræ (Fowler). As softening and excavation occur, the signs of cavity are added.

The *alimentary tract* becomes deranged, and anorexia and diarrhea are common complications. The latter symptom, occurring at the termination of the disease, indicates intestinal ulceration. Vomiting may be a troublesome symptom, resulting either from severe coughing paroxysms or from toxemia.

Prognosis.—The *prognosis* is unfavorable, especially when the disease develops at the period of puberty. Cases are no doubt arrested but it is impossible to foretell a relapse or a later complication, such as meningitis. If arrest in the stage of infiltration can be accomplished, the prognosis is more favorable. The constitution and family history must also be taken into consideration in forming an opinion as to prognosis. Much also depends upon the patient's environment and his economic condition. If the child can be sent to a favorable climate and receive every possible care the outlook is much brighter than for the poor city dweller.

Diagnosis.—A positive *diagnosis* is based upon a demonstration of the physical signs of infiltration and destruction of lung-tissue described above, associated with a persistent evening rise of temperature. Finding the tubercle bacillus in the sputum clinches the diagnosis. Early in the disease, however, it is not always possible to find unmistakable evidence of tuberculosis; and especially in children we are at a great disadvantage, owing to the difficulty of obtaining sputum for microscopical examination. Cough and emaciation in a child with a tuberculous family history, or with the history of having been exposed to such infection, together with slight evening pyrexia, are sufficient data to warrant a most thorough examination of the chest. The finding of localized subcrepitant rales at the apex of the lung, and prolongation of the expiratory sound in such a case, justifies the suspicion of tuberculosis. If sputum cannot be obtained a Roentgenogram should be made of the chest to confirm the diagnosis.

Treatment.—In the treatment of tuberculosis prophylaxis is to be considered first of all. Children with a tuberculous family history present an inherited predisposition to pulmonary tuberculosis. This predisposition is not, however, confined to such alone, as any constitutional enfeeblement in which the resistance of the organism is subnormal, especially when the chest is underdeveloped, offers a predisposing factor. Such

children should be brought up in a locality where the air is pure and uncontaminated, and they should be encouraged to lead an out-of-door life. The open-air school is a great boon to such children. Particular stress should be laid on the physical development of the chest by suitable and methodically carried out breathing-exercises and calisthenics; and for overcoming the cold-catching tendency, a cold sponge-bath, followed by brisk rubbing with a coarse towel, is most efficacious.

A careful inspection of the nose and throat should be made to determine the presence of local pathological conditions that may interfere with the child's breathing. The importance of the early recognizing of adenoids and enlarged tonsils, and their prompt removal when present, cannot be overestimated. Finally, in order to prevent infection, the child should not be permitted to come in intimate contact with a consumptive. When one of the parents is afflicted with open tuberculosis the strictest precautions should be taken on his or her part not to spread the infection to other members of the household.

Incipient cases are usually benefited by a change of *climate*. The requirements of a suitable climate are pure, uncontaminated air, equable temperature, and a maximum amount of sunshine. High altitude is by no means necessary; it best suits cases in which the disease is limited and there are no cavities. It may aggravate some cases by causing emphysema. Hemoptysis also contra-indicates a high altitude, and neurotic temperaments are aggravated thereby. A moderate altitude is preferable in most cases. The most suitable localities are the Adirondacks, the Southern pine regions, and the great plains bordering the Rocky Mountains. A location at sea-level seems better for chronic cases with emphysema, especially when there is nervous irritability, insomnia and loss of appetite. Many consumptives do not object to cold weather and are in fact benefited thereby. For these the Pocono Mountains, the Adirondacks and Colorado are good locations. Others do better in a warm, balmy climate such as the Southern Pine regions and Southern California.

The main advantage of climatic treatment, however, is the outdoor life invited thereby. No other form of treatment has yet given the promising results obtained in the sanatoria in which open-air treatment is systemically carried out, combined with forced feeding, rest, and judicious exercise.

When it is impossible to send the patient away he should receive all the benefits of the *open-air treatment at home*.

The *diet* is important. Patients that can be made to gain weight should not be despaired of. A change of climate often brings about a restoration of appetite when that has been on the wane, and may in this way alone confer great benefit. It is important to feed the patient as much as he can take; in fact, overfeeding has even proven beneficial in some instances. Milk and eggs are the chief foods for the tuberculous. Codliver oil is usually well borne by children, and is useful so long as it does not disturb the digestion. Even in the presence of pyrexia not above 100.4° F. we should not desist in our attempts at forced feeding.

Special Symptoms.—*Fever* calls for absolute rest in bed or the equivalent of the same. Sponge-baths should be used once or twice daily to reduce the temperature. The remedies most useful for the febrile symptoms are *baptisia*, *chininum arsenicosum*, *ferrum phos.*, and *iodine*.

Cough.—A cough which occurs in the morning and is accompanied by expectoration is useful, and should not be checked. Expectoration is materially aided by giving the patient a cup of hot milk, in the morning on awaking. On the other hand, a cough that continues during the night, causing loss of sleep, must be controlled (Fowler).

Codein is the most useful opiate for controlling cough but its use is usually undesirable. Among homeopathic remedies *hepar sulph.* 3x trit. is one of the most useful for the teasing night cough of phthisis. *Drosera* is highly recommended by Hughes (*Manual of Therapeutics*) for cough depending upon increased reflex excitability. *Belladonna*, *hyoscyamus*, *iodine*,

ipecac, *rumex crispus* and *tartar emetic* should also be considered. When profuse expectoration is present *stibium iodide* 2x (Goodno), *arsenicum iod.*, *lycopodium*, *stannum met.* and *calc. carb.* are the remedies most likely to prove useful. They must be carefully differentiated in order to prove helpful.

Hemoptysis, when slight and associated with hoarseness and tightness across the chest calls for *phosphorus*. Hughes places *phosphorus* foremost when the air-passages are much implicated in the morbid process.

Night-sweats are often difficult to control and are a source of great discomfort to the patient. They abate when the general condition is improved. Sometimes they are caused by too much bed covering and failure to carry out fresh air treatment to its full extent. There is therefore no advantage in using powerful therapeutic measures such as the administration of *atropine* to control these sweats. A cool sponge bath at bedtime is often beneficial. *China*, five drops of the tincture after meals and at bedtime is a good routine remedy. *Silicea* 6x trituration often acts very satisfactorily. *Iron* in the form of Blaud's pills or Ovoferin is often helpful when the patient is anemic. Hughes recommends *iodine* for nocturnal sweats. *Phosphoric acid* 3x will do a great deal for the debility resulting from sweats, diarrhea and bronchorrhea.

Gastric disorders may result from overfeeding. The best evidence of this is the presence of undigested food-particles in the stools (Fowler). When there is purely a gastric incompetency, *nux vomica* proves of great value. A catarrhal condition calls for such remedies as *pulsatilla*, *hydrastis* and *ipecac*. *Kreosote* is indicated when there is vomiting of glairy mucus, usually in the morning. It is a favorite remedy of the old school to improve the digestive function, increasing the appetite and checking flatulency.

The following list of remedies, with their clinical indications, should be studied for a fuller knowledge of the therapeutics of phthisis:

Aconite.—Pleuritic stitches, and blood-spitting after taking cold. *Ferrum phos.* is similar, but under this remedy there is less circulatory excitement, and anemia and vasomotor disturbances predominate.

Arsen. alb.—Dyspnea from exertion; cough between 1 A. M. and 3 A. M. Fever-heat and chilliness intermixed. Restlessness and thirst for small quantities of water. There is prostration and emaciation; anemia and edema of ankles; terminal diarrhea. Mostly indicated in the pneumonic type. *Arsen. jod.* 3x trit. freshly prepared is well suited to the fibro-caseous form of the disease when there is profuse purulent expectoration; emaciation; hectic fever and prostration. *Stibium iodide*, 2x trit., is highly recommended by Goodno in cases presenting profuse purulent expectoration. *Stannum iodide* has profuse purulent expectoration easily raised, and of sweetish taste. It is more useful in chronic bronchitis.

Baptisia.—Chill in forenoon or afternoon, followed by heat and perspiration; general weakness and languor. *Baptisia* is one of the best remedies for the pyrexia of phthisis, and has been extensively used since it was first recommended by Dr. J. S. Mitchell. It is usually employed in the tincture and lower dilutions.

Calc. carb.—"Pre-tubercular stage" in strumous subjects, the characteristic features being a form of indigestion associated with acid eructations and difficulty in assimilating fats (Hughes). Pale, rapidly-growing youths (*phos. acid*) or scrofulous children are especially benefited by this remedy. In the later stages it is indicated by tendency to perspire on slightest exertion; damp, cold feet; shortness of breath on ascending stairs; expectoration consisting of mucus with an admixture of pus which sinks in water, leaving the frothy mucus floating above.

China.—Septic fever, consisting of a chill, followed by high fever and sweat, usually occurring at regular intervals. Anorexia; chronic diarrhea. (Tincture and lower dilutions.)

The *arseniate of quinine*, 3x trit., is better indicated when the pyrexia is more irregular, especially if *arsenic* symptoms are present.

Ferrum phos.—Fever in the early stages, before septic infection has set in. Hemoptysis in the early stages not dependent upon excavation of lung-structure.

Hepar sulph., 3x trit., two grains every hour at night until cough is relieved. The cough is due to a persistent irritation in the larynx, not relieved by expectoration. It is excited by uncovering any part of the body, or by contact of body with cool bedclothes on first retiring. There is usually slight hoarseness, with rattling of mucus in larynx; however, expectoration does not relieve the symptoms. *Drosera* has a deep, spasmodic cough presenting this element of hyperesthesia, but there is not the free secretion present in *hepar*. *Hyoscyamus* has symptoms of cough worse on lying down at night; dry, spasmodic and titillating in character.

Iodine.—This remedy also presents characteristic cough symptoms. "Constant tickling in the windpipe and under the sternum, with expectoration of a transparent mucus, sometimes streaked with blood. Morbid hunger, soon after a meal, and yet loss of flesh. Dark hair and eyes" (C. G. R.). *Iodine* is one of the most useful remedies in pulmonary tuberculosis with active symptoms, namely, fever, cough, loss of weight.

Kali carb.—Sharp stitches in chest; cough worse 3 A. M.; puffiness of upper eyelids and swelling of ankles.

Lachesis.—Cough during sleep without awaking the patient; chilliness, followed by fever, with great talkativeness; sensation of suffocation; fluttering of heart; offensive stools.

Lycop.—Expectoration of large quantities of pus after unresolved pneumonia (C. G. R.). Cough day and night, the expectoration tasting salty. Hectic fever, with circumscribed redness of cheeks, usually late in afternoon (four P. M. to eight P. M., aggravation of symptoms). "It suits cases of a chronic and passive character, and is, I think, especially useful when phthisis occurs in young men."—(Hughes.)

Phosphorus.—Tormenting cough, often with hoarseness; worse toward midnight; tight and painful. There is tightness across upper portion of chest; inability to lie on left side. "Cough in the earlier stages of phthisis, with unusual implication of the air-passages in the morbid process." —(Hughes.)

Phosphoric acid is useful when the system has been drained by long-continued diarrhea or persistent night-sweats.

Sulphur.—Delayed resolution after pneumonia; chronic catarrhal deposits at apices, with a few moist rales. Neurasthenic individuals. Weak, gone feeling at 11 A. M., with craving for food or a stimulant. Morning diarrhea.

Tuberculin (Koch) has been successfully employed in bronchopneumonia, and is considered by Arnulphy capable of stopping the progress of incipient cases of tuberculosis of the lungs in a large proportion of cases (*Clinique*, June, 1897). *Avian tuberculin* is recommended by Cartier for suspicious bronchopneumonia. These nosodes have usually been given in the higher dilutions, either the 30th or 100th.

EMPHYSEMA.

Overdistention of the pulmonary air-vesicles occurs as a complication of most of the acute affections of the respiratory tract, resulting from either an interference with the function of a portion of the lungs (*vicarious* or *inspiratory emphysema*), or from an obstruction higher up in the tract, causing dilatation and even rupture of air-vesicles during expiration, especially when this is performed in a forcible manner. The latter variety, or *expiratory emphysema*, is the commonest and most typical form, occurring as a complication of whooping-cough, croup, asthma and measles. Chronic emphysema is occasionally seen in children as a result of chronic bronchitis and asthma.

Anatomically, emphysema is classified as *vesicular* or *alveolar*, and *interstitial*. In the latter form there is an escape of air into the connective-tissue stroma of the lungs, sometimes burrowing beneath the pleura and along the mediastinum into

the subcutaneous tissue of the supra-clavicular spaces. This results from trauma or from spontaneous rupture of a tuberculous area.

The *chronic* form, or *substantive emphysema*, is defined by Delafield as a chronic interstitial inflammation of the lungs, in which the dilatation of the air-spaces is a secondary phenomenon. Accordingly, it is a condition whose etiology and pathology are analogous to that of chronic endocarditis, endarteritis and nephritis.

In acute emphysema the upper lobes are principally affected, and most markedly in their anterior borders. In the chronic form both lungs are more or less affected in their entirety, but seldom to the extent observed in adults.

The *symptoms* of a compensatory emphysema are usually obscured by the original disease. Hyper-resonance, bulging of the supra-clavicular space during the expulsive efforts of coughing, exaggerated vesicular murmur and dyspnea are suggestive symptoms.

Chronic emphysema presents the typical barrel-chest; feeble respiratory murmur with prolonged expiration, diminished area of cardiac dulness; cyanosis, dyspnea, cough and expectoration; vesiculo-tympanitic percussion-note.

In the treatment of emphysema the underlying cause should receive principle attention. This is usually chronic bronchitis, asthma or cardio-renal disease.

Such remedies as *arsenicum*, *arsenicum iodide*, *aurum mur.*, *ipecac*, *lobelia* and *grindelia* are helpful for the respiratory embarrassment. *Coca* and *quebracho* are lauded by Hale as the only remedies giving continuous relief.

PLEURISY AND EMPYEMA.

Inflammation of the pleura is rarely encountered as a primary disease during childhood, but it is a frequent accompaniment of pneumonia. Pleurisy without exudation may accompany pulmonary disturbances of all kinds, and the

frequency with which adhesions and thickening of the pleural membranes are encountered in autopsies upon children points to the great prevalence of this condition.

The exudative variety of pleurisy in children is generally purulent in nature, and occurs most frequently as a complication of pneumonia, or develops simultaneously with the pneumonic process, in which case the condition runs the clinical course of a *pleuro-pneumonia* from the onset.

The acute infectious fevers are responsible for the development of some cases of pleurisy, and in older children a purely serous effusion may occur as a result of tuberculosis or it may be "rheumatic" in nature.

The micro-organisms playing the most prominent role in the etiology of purulent pleurisy are the *pneumococcus*, the *pyogenic micrococci* and the *bacillus tuberculosis*. *Pneumococcus pleurisy* is the most frequent form. It may occur simultaneously with a pneumonia, or develop as a sequel to the same (*meta-pneumonic pleurisy*). The exudate may be either sero-fibrinous or purulent. In the latter case the effusion is thick, creamy, and of a greenish color.

Streptococcus pleurisy is more common in adults. It is the type of pleurisy which usually complicates influenza and measles. The prognosis is not so favorable as in the pneumococcus variety. The course is more prolonged and the fluid re-accumulates after aspiration.

Tuberculous pleurisy may occur primarily, that is, in the absence of pulmonary tuberculosis; but in these cases tuberculosis of the bronchial glands is generally present. The effusion is sero-fibrinous at first and gradually becomes purulent. The course is slow and unfavorable.

Pathology.—In the early stages of a pleurisy the membrane appears injected and lustreless; later it becomes roughened and coated with a layer of fibrinous exudate. The extent of this process depends upon the severity of the attack, and it will vary from a delicate film of fibrin, coating only that portion of the

pleura directly covering the affected portion of lung in a pneumonia, to a general involvement of the entire pleural cavity, with a thick layer of inflammatory products over the lung and an abundance of purulent fluid in the pleural cavity. In these pronounced cases the pleura appears coated with a yellowish-green deposit of varying thickness; the opposing surfaces become adherent, forming pockets filled with pus. If serum is poured out freely during the first stage, adhesions do not occur, at least not to a great extent.

Symptoms.—A case of primary pleurisy begins with pain in the chest, gradually increasing fever and recurring chilly sensations. A painful, non-productive cough accompanies the condition. With the advent of the effusion the pain and cough become ameliorated.

The pain is expressed by severe crying after each coughing paroxysm or when the child is moved; there is also a tendency to avoid the affected side, and the respirations become abdominal in type and grunting in character. If the child be old enough to express its suffering, it may mislead us by referring the pain to the epigastric region or lower abdomen.

With the appearance of fluid, dyspnea develops, its severity depending upon the amount of fluid present.

The fever is remitting in character, seldom very high, rarely running above 103° F. As the acute symptoms subside the fever also falls but in the case of a purulent effusion there is a continued daily evening rise of temperature. Continued fever of obscure origin should suggest a sacculated empyema as a possible cause. Cases of pleurisy with effusion may clear up, suggesting that the effusion remained serous, but unfortunately the majority are purulent and require surgical interference.

When pleurisy develops *secondarily* during the course of a pneumonia or some other acute infectious disease there is usually the characteristic pain and an accession of fever to call attention to this complication. Sometimes, however, it is impossible to determine just when the pleurisy developed, and

the failure of a crisis to appear in a case of pneumonia together with the persistence of the signs of pulmonary consolidation are the first indications we may have of the associated pleurisy.

The physical signs by which pleurisy is recognized in children are mainly those indicating the presence of fluid in the thoracic cavity, as the early signs, namely, the friction-sound and localized pain, are not so readily elicited as in adults. By observing the posture of the child, however, and the fact that coughing induces severe pain, we are justified in suspecting the presence of an associated pleurisy. Conditions in which subcrepitant rales are present are a frequent source of error, they being easily mistaken for friction-sounds during infancy. For this reason the diagnosis of pleurisy depends upon a correct interpretation of painful respiration, painful cough, the characteristic onset and fever, and, still later, the demonstration of a pleuritic exudate.

In the early stages of pleurisy fixation of the thorax from the pain is often observed in children, producing a voluntary scoliosis, as pointed out by Ziemssen. As a result of this abnormal position, the ribs are brought closely together on the affected side and the percussion note becomes dull. Under these circumstances, therefore, dulness may be observed before exudation has actually set in.

After exudation occurs the symptoms are more characteristic. If the amount of fluid be considerable, there will be a noticeable bulging of the chest on the affected side, together with diminished motion. When the fluid occupies the left pleural cavity the heart is displaced to the right; when occupying the right pleural cavity there is a downward displacement of the liver. The pleural fold is also displaced beyond the midsternal line.

Vocal fremitus is absent over the site of the fluid, while the percussion-note is flat and there is increased resistance. These two signs are among the most important data in the diagnosis of effusion. Above the level of the fluid tympanitic

resonance is obtained when the lungs are not entirely deprived of air. The line of flatness will change its direction with a change in the position of the patient, providing the fluid is not inclosed by adhesions.

In recent cases bronchial breathing is very frequently heard above the line of dulness, together with Skodaic tympany to percussion. At the upper level of the fluid the spoken voice has a characteristic nasal twang (egophony). The breath sounds over the fluid may be bronchial in character. It is only in cases of large effusion that the breath sounds entirely disappear. Vocal fremitus is always diminished, however, over the fluid and the line of dulness is higher in the axilla than anteriorly or posteriorly and does not follow the outline of a pulmonary lobe as in pneumonia.

In children under three years the fluid is almost invariably purulent, and even until puberty there is a predisposition to empyema. According to Baccelli a purulent exudate is less likely to transmit the whispered voice, but this is not always the case. Subcutaneous edema of the thorax on the affected side is a late manifestation of empyema. A positive diagnosis cannot, however, be made without the use of the aspirating needle, which is perfectly safe when used under proper aseptic precautions. In old cases, where the pus is too thick to be drawn into the needle, even this method will lead to error unless the negative result is properly interpreted. In a serous exudate, the presence of chain cocci, staphylococci, or the diplococcus pneumoniae, indicates that it will become purulent (Koplik). Tuberculous pleurisy is recognized by finding the tubercle bacillus in the effusion and according to Dieulafoy by the exclusive presence of lymphocytes and red blood corpuscles. In the other forms of infections pleurisy polynuclear and large mononuclear leucocytes predominate.

Diagnosis.—The early diagnosis of fluid in the chest is of the utmost importance, particularly as the recovery of the patient depends largely upon the time when proper treatment

has been instituted. Many difficulties may be encountered in establishing a diagnosis, especially as the effusion is not generally a large one and because it is usually secondary to pneumonia—*metapneumonic pleurisy*. The history is therefore not as clear as in primary pleurisy. Again, owing to the marked tendency for the fluid to become encapsulated, it does not produce the characteristic physical signs expected of free fluid in the chest. The determination of the character of the fluid has been fully discussed above.

The chief indications upon which the diagnosis can be made are absence of vocal fremitus; flat quality of the percussion-note and resistance; bronchial breathing and bronchophony over the entire affected side posteriorly and displacement of viscera. Koplik lays special stress upon displacement of the pleural fold and personally I have found it a most important physical sign. Normally these folds meet in the midsternal line and when there is considerable fluid in either side of the chest cavity dullness will be found to extend beyond the median line over toward the well side. In smaller effusions auscultatory signs are not characteristic and may be misleading on account of the good conduction of sound in the child's chest. Dullness, with a sense of resistance to the finger and absence of vocal fremitus are the signs to be relied upon in such cases.

Empyema should always be suspected when the temperature remains high for a period beyond two weeks in cases of pneumonia, especially when bronchial breathing persists.

Encapsulated fluid in unusual sites, such as the upper portion of the chest, is very difficult to differentiate from persistent bronchopneumonia and abscess of the lung. In the latter condition percussion and auscultation give practically the same signs, but the presence of loud, coarse pleuritic friction sounds are of importance as favoring the diagnosis of abscess (Holt, *Archives of Pediatrics*, Jan., 1904).

Pericardial effusion must also be borne in mind as a possible condition likely to be confused with sacculated empyema.

Prognosis.—Serous effusions are usually absorbed readily, seldom persisting over three weeks. If however pus producing micro-organisms gain entrance into the pleural cavity the prognosis is changed. As stated above, an empyema due to the pneumococcus presents the most favorable prognosis, although it may run a prolonged and tedious course. When the streptococcus is present open drainage offers the only chance for a cure. The tuberculous variety is the least favorable. Spontaneous evacuation through the chest wall (usually in the region of the fourth or fifth rib) or through the bronchial tubes, by perforation into the lung parenchyma may take place in any case of untreated empyema. At times perforation into the peritoneal cavity takes place. The usual cause of death in an untreated empyema is the gradual exhaustion or amyloid degeneration accompanying prolonged suppuration, or one of the above mentioned accidents.

When the fluid is removed early there is a fair chance for the compressed lung being restored to complete function; on the other hand, if the condition has been one of long standing, dense bands of adhesions are formed to such an extent that the lungs become crippled and the thorax deformed.

Treatment.—Hot applications are helpful in the early stages. Strapping the chest is not advisable in cases of secondary pleurisy. Fluid which is present in considerable amount should be promptly evacuated if absorption is not progressing rapidly; under no circumstances should accumulations of fluid be allowed to remain in the chest for a period exceeding two weeks, unless decided improvement is noted daily. If the fluid is purulent it should be withdrawn every second or third day during the acute stage of the disease. Radical surgical treatment should not be instituted until the associated pneumonia has run its course and the toxemia begins to subside. The operation of choice in infants is simple incision in an intercostal space with the introduction of a drainage tube while in older children the resection of a piece of rib is perfectly safe and gives the best results.

Diagnostic puncture of the chest is most satisfactorily performed with a Luer syringe. The usual site of puncture is the sixth or seventh interspace in the axillary line or the eighth interspace posteriorly. The needle should not be inserted too close to the spine, and should be directed toward the upper border of the rib rather than to its lower, on account of the intercostal arteries. Koplik insists on puncturing at the site indicating fluid, as elicited by flatness and absence of vocal fremitus, rather than at some point of election; when the empyema is localized this rule is absolutely essential to follow.

Remedies.—*Aconite*, *arnica*, *belladonna*, *bryonia*, *kali carb.*, *rhus tox.*, and *scilla* are indicated in the early stage of pleurisy.

When exudation is abundant, *apis*, *cantharis*, *iodium* and *Sulphur* are most frequently indicated.

The clearing up of a purulent exudate, after proper surgical measures have been instituted, is helped by *hepar sulphur*.

Acon.—Sharp, stitching pain in side; high fever, restlessness and chills; after exposure to cold, dry winds or checked perspiration.

Apis—Pleuritic effusion; scanty urine.

Arnica.—Traumatic cases; hemorrhagic effusion.

Arsenicum.—Profuse serous effusion; dyspnea; cachexia; prostration; empyema. The *iodide of arsenic* is well suited to tuberculous cases, as is also *iodium*.

Bellad.—Cerebral symptoms; complicating the infectious fevers or exanthemata.

Bryonia.—Early stage of all pleurisies, and in dry pleurisy frequently to the end. *Sulphur* is needed in the latter cases to complete the cure. Sharp, stitching pains, aggravated by motion and deep breathing; friction sounds and local tenderness.

Canth.—Profuse serous exudation; frequent cough; dyspnea; palpitation; profuse sweats; great weakness; tendency to syncope; scanty and albuminous urine.—(E. Faivre.)

Colchicum.—Rheumatic diathesis; sour-smelling sweats; scanty red, turbid urine, with abundant uric acid and some albumin.

Hepar.—After drainage has been established. Profuse, purulent discharge from pleural cavity.

Kali carb.—Violent stitching pains, especially on left side, worse in early morning (after fresh adhesions have formed during sleep), accompanied by dry cough and palpitation of the heart. When *bryonia* fails to give relief.

Iodium.—Tuberculous pleurisy.

Mercurius.—Syphilitic or rheumatic diathesis; pains persisting after the fever subsides; constant chilliness, with tendency to sweat; gastro-intestinal catarrh; perihepatitis. *Merc. corr.* is useful in pleuritic effusions accompanying parenchymatous nephritis.

Rhus tox.—Acute rheumatic cases, after exposure to wet. General aching and prostration; continued fever with great restlessness; typhoid state.

Scilla.—Sharp stitching pains in side with broncho-pneumonia; prostration; cardiac weakness. Cannot lie on left side.

Sulphur.—Stage of effusion in primary serous pleurisy. *Sulphur* is the best remedy to hasten the absorption of a non-purulent pleural effusion.

CHAPTER X.

DISEASES OF THE HEART AND ITS MEMBRANES.

Organic heart disease in childhood may be either congenital or acquired. Congenital affections result from fetal endocarditis or from developmental defects. Acquired heart disease in childhood is the result of infection (rheumatic) and presents the same pathological processes observed in adults with the exception that the degenerative and luetic changes affecting especially the aortic valves and the bloodvessels in adult life are not encountered in childhood. Functional disorders are rare and of little clinical significance. Sinus irregularity and neurotic pains or palpitation are the chief functional disturbances encountered.

The heart is relatively larger in infancy than in later life, but it does not increase in size proportionally with the growth of the child, developing only slightly during the first five years of childhood (Barthez and Rilliet). It occupies a higher and more horizontal position than in the adult, and for this reason cardiac dulness extends relatively further both to the right and to the left of the sternum. At the sixth year dulness may still extend beyond the right border of the sternum, and the apex is generally found outside of the left nipple-line up to the fourth year. The apex may be in the fourth intercostal space until the sixth or seventh year. After the seventh year, however, it should be located within the left nipple-line and in the fifth intercostal space. It is important to remember that the nipple is not an absolutely fixed point, and that it may be found in the third or fourth intercostal space or over the fourth rib. Most frequently it is situated over the fourth rib, somewhat nearer the median than to the mid-axillary line.

In young children deep cardiac dulness extends beyond the left mammary line and on the right it may reach the parasternal

line. After the seventh year the position of the apex and the area of deep dulness are relatively the same as in the adult.

Owing to the yielding character of the sternum and of the costal cartilages, enlargement of the heart may cause a decided bulging of the front of the thorax up to the third year. This is usually seen in congenital heart disease. The third piece of the sternum may be misplaced at even a later period, as Rotch pointed out, owing to the fact that it is ossified later than the upper portions. Pericardial effusion will cause bulging in the regions of the heart at any period of childhood. The figures obtained by measuring the distance from the midsternal line to the outer edge of the apex furnish valuable data for estimating the extent of cardiac enlargement, and are also an important matter of record for future reference. From the examination of a large number of normal children at different ages I have obtained the following data: Distance from the midsternal line to the outer edge of the apex in the newborn, 4.5 cm. to 5 cm. By the tenth year it is 7.5 cm., or 3 inches, in the average case. In a male child from one to two years old it is from 5.5 to 6 cm., usually a trifle less in females. From the fourth to the sixth year it averages 6 to 6.5 cm. and may reach 7 cm. by the seventh year. In severe forms of valvular heart disease it is not uncommon to find the left border reaching from 11 to 12 cm. from the midsternal line. The yearly gain in the distance of the apex from the median line seems trifling and does not appear to correspond with the increase in the size of the heart, but it must be remembered that the heart is relatively large in early childhood and also that it assumes a more vertical position with the fuller development of the child.

The *pulse* is soft and dicrotic in character during childhood; it is rapid and irregular in infants. Its rate is about 130 at birth; 120 at the end of the first year, and usually remains about 100 up to the fifth year. The blood pressure is normally low. A rapid pulse may be of purely nervous origin and is

therefore not a trustworthy sign of cardiac weakness. Arrhythmia is also of little significance as it is usually of the type of sinus irregularity. A slow pulse however is more significant. This is characteristically seen in diphtheritic myocarditis.

Heart sounds.—The first sound of the heart obtained over the apex is the loudest and has the characteristic booming quality heard in the adult. During infancy, however, the muscular element is lacking and the first sound is short and more distinctly valvular in quality. Throughout the entire period of childhood the pulmonary second sound predominates over the aortic second sound. In valvular heart disease the pulmonary second sound becomes distinctly accentuated. Heart murmurs are common in childhood and it is important to determine whether they signify an organic lesion or whether they are functional or purely accidental.

Anemic or Functional Murmurs may be heard in a severe case of anemia at any time of life. They are most commonly encountered in older children suffering with chlorosis. They are heard loudest at the pulmonary valve, and are systolic. There is no heaving impulse, accentuated second sound, or extension of the apex-beat beyond the mammillary line. The pulmonary area is so frequently the seat of murmurs that Balfour has referred to it as the area of auscultatory romance.

Cardio-pulmonary murmurs (Hochsinger) are produced by the transmission of the contractions of the heart and its movements to the lungs. These murmurs are systolic, and are differentiated from anemic murmurs by their definite relation to the respiratory function, being increased during forced and suspended by a cessation of respiration. They are common in children with deformed chests, due to rickets or Pott's disease, and are best heard over the precordial region.

The endocardial systolic murmur is an indication of a leakage at the mitral valve. In the active stage of the endocarditis there is fever, evidence of heart weakness, increase in the area of cardiac dulness and accentuation of the pulmonary second

sound. After the endocarditis has subsided, mitral regurgitation may remain as a permanent defect.

Other murmurs which may be heard in chronic valvular disease are the *presystolic*, which may also be felt as a thrill in the precordial region. The presystolic murmur is fugitive in character and may only be heard when the patient has been active, disappearing on resting.

CONGENITAL DISEASES AND DEFORMITIES.

Congenital defects such as nonclosure of the foramen ovale or a patent ductus arteriosus may exist without producing clinical symptoms. A lesion of the pulmonary artery, however, produces unmistakable signs of circulatory obstruction. This lesion is seldom found existing alone, for the increased intraventricular pressure which results from the pulmonary stenosis leads to a nonclosure of the auricular and ventricular septa or to a dilatation and persistence of the ductus arteriosus. Clinically, therefore, a combination of a lesion and a defect is the common finding. In Holt's series of 242 cases the following combinations were the ones most frequently encountered, in the order given: Pulmonic stenosis with defective auricular septum, the three lesions associated, or the first two with a patent ductus arteriosus.

Inflammation of the endocardium in the fetus is of the chronic or sclerotic variety, verrucose endocarditis being very rare (Osler). Small, nodular bodies, the remains of fetal structure (Bernays), and small, rounded, bead-like bodies of a deep purple color, which are the remains of a hemorrhage (Osler) have frequently been mistaken as evidences of endocarditis, leading to a misconception as to the prevalence of this affection. The belief in fetal endocarditis has been abandoned by some of the more recent investigators in this field and even pulmonary stenosis is classed by them as a developmental defect.

Defects of the ventricular septum is most frequently associated with pulmonic stenosis or defect of the auricular septum.

The defect is most frequently found in the anterior muscular portion of the septum (Rokitansky). If compensatory hypertrophy of the right ventricle supervenes, no apparent symptoms may be present.

Patency of the foramen ovale may exist without any evidence of cardiac disease. When, however, other anomalies increasing the pressure in the right auricle co-exist, a mixing of venous and arterial blood takes place, with resulting cyanosis.

Stenosis of the pulmonary artery is the most important congenital affection, the above mentioned conditions in the majority of instances being the direct results of the pulmonary stenosis. The symptoms depend upon the amount of constriction at the pulmonary orifice. The infant may die shortly after birth with intense cyanosis and asphyxia, or it may grow up to adult life, with, however, defective blood aeration; cyanosis, usually manifest when crying or after physical exertion; coldness of the extremities, clubbing of the finger-nails, and mental and physical backwardness. Uncomplicated, and therefore uncompensated, pulmonary stenosis usually leads to death in early infancy.

Patent ductus arteriosus does not necessarily produce symptoms. When, however, the ductus is dilated as a result of an associated lesion we may suspect this condition from the presence of a thrill at the base of the heart and the transmission of a murmur into the carotids. It also induces hypertrophy.

Abnormalities in the origin of the great vessels are rare, and lead to early death or make extra-uterine life impossible, unless there is an open foramen ovale or a communication between the pulmonary veins and the right side of the heart.

Tricuspid insufficiency and stenosis are grave defects, resulting from endocarditis. There may be complete artesia of the orifice, in which case a degree of circulation is maintained through an incomplete ventricular septum. The right heart becomes dilated and hypertrophied; there is cyanosis and tendency to venous hemorrhages.

Affections of the *left heart* are exceedingly rare and usually masked by other defects.

Symptoms.—The most characteristic symptom of congenital heart disease, and the one which, as a rule, first calls our attention to the fact that the infant is afflicted with a cardiac defect is cyanosis. The baby is a “blue baby,” either showing some degree of cyanosis continuously, especially at the finger tips, the lip and toes, or it becomes cyanosed when crying. Cyanosis is usually detected soon after birth; sometimes it is latent and first observed during an attack of bronchitis or during a severe crying spell.

Cyanosis is absent in defective ventricular septum and may be absent in cases with patent ductus arteriosus. Well defined cyanosis always indicates pulmonary stenosis. When cyanosis is absent or only present to a slight degree congenital heart disease may not be suspected until a thrill or murmur is detected during a routine examination of the infant. The remote effects of congenital heart disease are a stunting of the growth of the child; clubbing of the finger tips and toes; dyspnea on exertion; susceptibility to pulmonary affections. Cases without cyanosis may attain adult life and enjoy apparently good health while those with marked cyanosis suffer with malnutrition and die in early childhood. Intermediate cases present about the same problems as the child with acquired heart disease.

The *diagnosis* rests upon a recognition of the above mentioned symptoms, together with the associated physical signs.

Stenosis of the pulmonary artery presents hypertrophied right heart; loud systolic murmur over the second and third costal cartilages to the left of the sternum, not transmitted into the carotids, and a thrill. The pulmonary second sound is weakened. When these signs are present in an infant past its first year it may be assumed that the foramen ovale has remained patent or there is an associated septum defect. When there is a loud, buzzing murmur transmitted into the carotids

and subelavians, together with accentuated pulmonary second sound and hypertrophy of both ventricles, there is probably associated an open ductus arteriosus (Hochsinger, *Auscultation des Kindlichen Herzens*).

Patency of the ductus arteriosus leads to hypertrophy of the right ventricle and dilatation of the pulmonary artery. The characteristic murmur is a continuous humming sound transmitted into the carotids.

The following table is given as an aid in the classification and differential diagnosis of the different forms of congenital heart disease:

1. Cases *with cyanosis*: Pulmonary stenosis.
2. Cases *without cyanosis*: Patent foramen ovale; defect of ventricular septum; open ductus arteriosus.
3. Cases *with hypertrophy*: Pulmonary stenosis, right ventricle; patent ductus arteriosus, right ventricle and pulmonary artery; defect of ventricular septum, both ventricles.
4. Cases *without hypertrophy*: Open foramen ovale.

Murmurs.

Systolic murmur and thrill over pulmonary area—pulmonary stenosis.

Systolic murmur at apex, not transmitted to carotids—defect of ventricular septum.

Systolic or humming-top murmur, transmitted into carotids—open ductus arteriosus alone.

Systolic murmur transmitted to carotids with thrill, cyanosis and hypertrophy—Pulmonary stenosis with associated open ductus arteriosus.

The *treatment* is largely constitutional. The feeding of these cases usually presents difficulties and is to be carried out along the lines as suggested for the feeding of difficult cases. Protection of the child against exposure and against the acute infectious diseases is imperative. Acute affections of the respiratory tract are especially to be feared. Attacks of cyanosis

or threatened cardiac failure and dyspnea will call for stimulation with either aromatic spirits of ammonia or brandy.

Cases without cyanosis present a better prognosis than those in whom this symptom is present. They should not be restricted too much in their play as a certain amount of exercise is beneficial to them. Many cases with a congenital lesion, notably an open ductus arteriosus, attain adult life and are but slightly handicapped by their affliction.

On general lines *aconite*, *arsenicum*, *camphor*, *cuprum*, *digitalis*, *glonoin*, *lachesis*, *rhus tox.* and *veratrum viride* are to be considered, their symptomatology covering the conditions met with in these cases, namely, hypertrophy, dyspnea, excessive heart-action, cyanosis, etc.

PERICARDITIS.

Pericarditis, or inflammation of the pericardium, may be either acute or chronic and primary or secondary. Acute pericarditis in infancy usually occurs as a complication of pneumonia, notably pleuro-pneumonia. In older children it is most frequently a complication of rheumatic fever, in fact, involvement of the pericardium is to be anticipated in all severe cases of rheumatic carditis. Acute pericarditis may also occur as a complication of scarlet fever, focal infection and sepsis. It may occur from traumatism and from the extension of the infection from the lung or pleura. A chronic, adhesive type of pericarditis which is probably tubercular in origin is occasionally encountered in which there is associated involvement of other serous membranes. The name "*polyserositis*, or *multiple serositis*" is given to this distinct clinical form of pericarditis.

The *pathological changes* in pericarditis are the same as are observed in inflammation of other serous membranes. In the dry form, there is merely a loss of the normal gloss and smoothness of the membrane covering the heart and lining the pericardial sac together with the deposit of fibrinous exudate. The process usually begins at the base of the heart where the inflam-

matory reaction is most marked and where the first friction sounds are generally heard. In the serous variety an abundance of serous fluid is poured out which distends the pericardial sac causing a bulging in the cardiac area and a muffling of the heart sounds. The fluid usually absorbs spontaneously, leaving, however, a roughened membrane covered with exudate with resulting adhesions between the heart and pericardium. Adhesions and obliteration of the pericardial sac are however more likely to occur in the dry, or fibrinous than in the serous form.

Purulent pericarditis does not undergo spontaneous absorption and presents a most unfavorable prognosis. It is a frequent complication of fatal cases of pleuro-pneumonia and of sepsis.

Chronic pericarditis results either from recurring attacks of rheumatic pericarditis or it is tubercular in origin. The tubercular cases rarely show tubercles upon the pericardial surface; the pathological changes observed are similar to those seen in chronic, proliferating pleurisy. There is usually involvement of the tissues of the mediastinum causing dulness in the upper sternal region. Chronic mediastinitis, either tubercular or rheumatic, causes physical signs often mistaken for aortic disease. Chronic rheumatic pericarditis is almost invariably associated with valvular heart disease while the tubercular form complicates pulmonary tuberculosis or is one of the lesions of a polyserositis.

Symptoms.—Pericarditis is rarely recognized in infants, being usually a complication of such a condition as pleuro-pneumonia or sepsis.

If the child is old enough to complain of pain in the region of the heart, which may also be referred to as radiating to the left shoulder or epigastrium, or as occurring alone in these locations, a careful physical examination may reveal local tenderness and possibly cardiac friction-sounds over the base of the heart. If friction-sounds are elicited, they will be heard as rubbing or crackling sounds synchronous with the heart's action and independent of respiration. They are loudest under the

fourth rib to the left of the sternum, and may simulate a mitral regurgitation murmur. The loud friction-sound disappears when fluid accumulates in the pericardial sac but there is usually a persistence of some friction at the base, probably due to an associated mediastinitis.

With the appearance of the effusion the pulse becomes feeble and irregular. Oppression, dyspnea and cyanosis develop with the outpouring of sufficient fluid to embarrass the heart's action; and eventually convulsions, and in older children delirium and coma, close the scene in fatal cases. A rapid outpouring of serum into the pericardium may produce sudden death. We sometimes see this occur during an attack of rheumatic fever and in pneumonia.

Bulging of the precordial region, increased area of cardiac dulness, and muffling of the heart sounds are the characteristic physical signs of pericardial effusion. The area of dulness is not triangular as in adults, and the heart, with its distended sack, retains its normal position, simply enlarging. Enlargement is more pronounced to the left. Unless dulness reaches up to the second interspace on the right side, it is more likely due to dilatation of the right ventricle than to fluid (Koplik). The percussion note is flat and resistant.

Adhesions are to be suspected when there is a displacement of the apex not due to marked hypertrophy, or cardiac dilatation and retraction of the intercostal space during systole. The mere retraction of the apex region during systole is by no means diagnostic of pericardial adhesions. When, however, this is associated with retraction of a considerable area of the thorax during systole, which rapidly returns to normal during diastole, we have strong evidence of the same (Gerhardt, *Lehrbuch der Auskultation u. Percussion*). Perhaps the most conclusive sign is that pointed out by Broadbent, namely, retraction of the lower intercostal spaces posteriorly, due to tugging on the adherent diaphragm. Broadbent's sign is conclusive evidence of extensive pericardial adhesions but is rarely encountered.

The only definite physical sign upon which a positive diagnosis can be made is the fixed position of the heart irrespective of the patient. This is demonstrated by noting the position of the apex beat and deep cardiac dulness with the patient standing, recumbent and lying on the right side. Together with this sign there is progressive asystole rebellious to any form of treatment (Apert, *Maladies des Enfants*). When systolic retraction in the precordial region is associated with diffuse diastolic collapse of the jugular veins the probabilities of an adherent pericardium are increased.

The *prognosis* of pericarditis is always grave, particularly when complicating pneumonia and scarlet fever. A serous, rheumatic pericarditis usually undergoes spontaneous absorption but the danger of pericardial adhesions and recurrence must always be considered. The prognosis of adherent pericardium is bad because of the secondary changes in the heart muscle which result therefrom.

Treatment.—Absolute rest in bed is imperative until every evidence of fluid or cardiac weakness has disappeared. With large effusions aspiration may become necessary; purulent pericarditis requires surgical treatment.

Acon.—Chilliness; hard, bounding pulse; sharp pain in region of heart; great restlessness, dyspnea and nervous excitement.

Arsen.—Great anguish and oppression; constantly changing position; cyanosis; thirst; in consequence of repelled exanthems, or in connection with pneumonia; stage of effusion.

Bryonia.—This remedy follows well after *aconite*, and is most applicable during the stage of effusion, although it seldom absorbs the exudate completely. *Sulphur* is a most valuable remedy for this purpose, especially when the case becomes protracted.

Cactus grand.—Sensation of constriction about the heart, as if a strong hand were grasping it. There may also be a sense of deep-seated soreness in the precordium, with dyspnea; attacks of suffocation; fainting, small, irregular pulse.

Digitalis.—Copious serous effusion; small, irregular, rapid pulse; diarrhea and vomiting.

Iodium.—Complicating croupous pneumonia. Violent palpitation and oppression from slightest motion; must lie perfectly quiet on back.

Spigelia.—After *aconite*, when the friction sound becomes audible. Sharp, stitching pains in chest. *Spigelia* is a most efficient remedy for the painful stage.

ENDOCARDITIS; VALVULAR HEART DISEASE.

Acute endocarditis in childhood is most frequently a complication of rheumatism and may occur as the primary manifestation of this affection. Joint symptoms are less characteristic of rheumatism in children than in adults but endocarditis is more likely to develop as a complication of this disease in childhood. Chorea, which is one of the clinical manifestations of rheumatism in childhood, is also frequently complicated with endocarditis. Every case of rheumatism, therefore, however mild the joint manifestation may be, and every case of chorea, should be watched for signs of endocarditis. Endocarditis may also occur as a complication of tonsillitis, scarlet fever, pneumonia, gonorrhea and septicemia. There is a special type of endocarditis resulting from a general blood infection with the streptococcus mitis which is described as septic endocarditis. This form of endocarditis is far less common than the rheumatic variety. The pathological process in bacterial endocarditis is a destructive one (ulcerative endocarditis) and the prognosis is most unfavorable.

In simple, or verrucose endocarditis the valves are covered with inflammatory excrescences—endocardial vegetations. These result from the deposit of fibrin and leucocytes upon the necrotic areas which are produced in the endocardium by the bacteria and their toxins. The valves become thickened, shortened and distorted and consequently incompetent. The mitral valve is the one chiefly involved. In cases of bacterial, or septic endo-

carditis, both the mitral and aortic valves are usually affected. Portions of the valvular deposit may become detached and swept into the general circulation, producing emboli at distant points.

Chronic endocarditis, or *chronic valvular heart disease* is a sequel of acute endocarditis. In children there is a notable tendency to recurrence of the endocardial inflammation, each new attack increasing the disability of the already damaged valves and thus adding to the strain on the heart muscle. The commonest valvular defect resulting from these pathological changes is *mitral regurgitation*, although *mitral stenosis* and *aortic regurgitation* may also be encountered.

Symptoms.—An irregular continued fever in childhood, at times hardly perceptible, often disappearing after rest in bed and accompanied by malaise, pallor and moderate anemia should always arouse the suspicion of a rheumatic infection. In the absence of active throat manifestations or of joint involvement this febrile disturbance should at once lead us to suspect the presence of a low grade endocarditis. Tuberculosis, of course, must be excluded as a possible cause for such a rise of temperature. At the onset of endocarditis repeated examination of the heart will reveal a weakening and blurring of the first sound at the apex with the gradual development of a blowing murmur. With the establishment of the murmur the area of deep cardiac dulness increases and the pulmonary second sound becomes accentuated. These signs are found in the great majority of cases because the mitral valve is the one most frequently attacked by rheumatic endocarditis and a mitral insufficiency is the commonest lesion resulting therefrom. In the early stages the murmur may be heard more distinctly just over the area of the auriculoventricular orifice than at the apex, and owing to the adaptability of the child's heart to any extra strain the pulmonary second sound may remain unaltered for some time. The murmur just described may also occur in the myocarditis associated with diphtheria and other acute infectious diseases. However, in such cases, it is only of temporary

duration and disappears during convalescence from these diseases after the heart muscle has regained its tone.

Mitral stenosis may develop in conjunction with insufficiency or exist alone. The first physical sign to be noted is a faint murmur occurring in early diastole. The cause of this murmur is the flow of the blood current over the roughened edges of the auriculo ventricular orifice during the early part of diastole. Later, as contraction and distortion of the orifice develop the auricle is called upon to force the blood through the stenosed orifice, and as a result of this exaggerated auricular contraction in late diastole we now hear the loud rumbling presystolic murmur accompanied by a palpable thrill, a sharp snapping first sound and usually reduplication of the second sound. The last results from the high tension in the pulmonary artery.

Aortic disease is rare in childhood and is indicative of a severe progressive type of endocarditis (*subacute bacterial endocarditis*). At first a systolic murmur over the aortic area transmitted into the carotid and accompanied by a weakening of the aortic second sound is heard. The cardiac enlargement progresses and the patient's discomfort is much increased. This is followed by throbbing carotids, collapsing pulse and total obliteration of the aortic second sound showing that the valves have become incompetent. In the general cardiac turmoil an aortic diastolic sound is difficult to detect and this is unnecessary for the diagnosis.

The severe cases of acute endocarditis which one is apt to encounter usually present the following history: A child from five to ten years is attacked with sore throat followed by arthritic manifestations of moderate severity. There is slight fever. Suddenly the temperature rises, the patient complains of pain in the cardiac region and epigastrium; there is cough and shortness of breath. Examination of the heart reveals increase in the area of cardiac dulness, a loud murmur at the apex and possibly friction sounds near the sternum. Circulatory failure progresses until a fatal termination sets in. The duration of the entire illness may be but a few weeks.

More common, however, is the recurring form and this furnishes the cases we see most frequently in the hospital wards. The child is brought to the hospital for an attack of rheumatism, chorea, or for shortness of breath, and an old valvular lesion is discovered. Inquiry into the past history of the case discloses the fact that the child has had a previous attack of rheumatism or chorea, or perhaps such vague symptoms as sore throat and growing pains. The rheumatism or chorea subsides, but the fever continues, due to the presence of endocarditis. After several weeks in bed the process subsides, the heart muscle regains its tone and the breathing and pulse improve. The child is now dismissed from the hospital, but we anticipate his return in the near future in a worse condition than on the previous admission. Fully half of these cases succumb to their malady before puberty.

Prognosis.—The literature upon heart disease in childhood agrees more or less in giving to the prognosis an unfavorable aspect. It is, however, difficult to get a clear conception of this important question because of the lack of definite statements that are to be found. The statistics given by Dunn (*American Journal Diseases of Children*, August, 1913), have furnished much valuable information in this direction.

In the first place, according to Dunn's figures, the ultimate disability following rheumatic heart disease acquired in early childhood is not nearly so great as that following endocarditis occurring in late childhood. Thus, out of a series of 88 cases in young adults with evidence of former endocarditis only two showed great disability and in these the attack occurred in the twelfth and thirteenth year. The younger the child, therefore, when the endocarditis occurs, the better the chance for the future as far as permanent after effects are concerned. Compensation is established not only by means of a mechanical hypertrophy of the heart but also through mutual adaptation between the heart and the organism.

These figures, however, should not lead us to overlook the

real and more important side of the question, namely, that it is only the mild, non-recurring cases that live beyond puberty and make up this class of young adults with a non-serious well compensated valvular lesion. The *immediate mortality* out of a series of 261 of Dunn's cases was 20 per cent. Out of the remaining 209 cases 50 per cent died during the subsequent ten years.

From these figures, then, it may be seen that the majority of children attacked with endocarditis do not live beyond puberty. One of the chief reasons for this unfortunate outcome is the liability to recurrence which is so strong in rheumatic fever, being noted in fully 80 per cent of cases. With these recurrences increase in the existing danger to the heart occurs. Another factor, already mentioned, is the tendency to a general carditis, the process not remaining limited to the lining of the heart as in the adult. We must also take into consideration the unusual demands made upon the heart of the young growing organism in order to maintain an adequate circulation of the blood.

Even in spite of these demands and in the presence of a badly damaged valve, the child's heart seldom fails and heart failure in childhood usually results from an acute process or from an acute exacerbation of an old condition. During such an attack, fever, cough, shortness of breath and pain are the prominent symptoms and the child often dies before edema of the extremities and enlargement of the liver develop. *Reinfection and not strain*, as in the case of adults, leads to the final breakdown. It is perhaps chiefly for this reason that remedial measures that so frequently relieve the symptoms of decompensation in the adult are futile in these crises in childhood.

The worst prognosis may be anticipated when pericarditis occurs as a complication. A progressive asystole, rebellious to any form of treatment, may be looked upon as the chief clinical symptom of an adherent pericardium (Apert, *Maladies des Enfants*). Acute pericarditis with effusion gives a high immed-

iate mortality (31 per cent, Dunn). An adherent pericardium leads to progressive heart failure; ascites and anasarca may become prominent features and the case assume the clinical type of pseudo-cirrhosis of the liver.

To recapitulate, the child's heart if not too seriously involved and if not handicapped by recurring infections may hypertrophy in a most favorable manner and adapt itself to a valvular defect; in fact, "the entire organism adapts itself to the new condition arising in the circulatory apparatus" (Feer, *Kinderheilkunde*). Unfortunately this occurs in the minority of instances. We occasionally meet such cases in our practice and follow them through puberty to early adult life, but always with misgivings.

A fortunate circumstance in connection with rheumatic fever is that after adult life is reached there is less tendency to endocarditis than in childhood.

The chief reasons for the unfavorable outlook in rheumatic heart disease in childhood is the tendency to recur and the frequency with which myocarditis and pericarditis are associated with the endocarditis.

Cardiac insufficiency in children results from infection or reinfection, and not from strain, as does the ruptured compensation in adults. The particular valve involved is of no special prognostic significance.

Involvement of the aortic valve is grave, for the reason that it is practically never affected alone, but only in conjunction with the mitral valve in the more violent types of endocarditis. Many of these cases assume the type of "septic endocarditis."

Diagnosis.—Cases of valvular heart disease may exist for a long time unrecognized, and be first discovered during a routine examination of the chest. This is especially so when the endocarditis has developed insidiously during an attack of chorea or in conjunction with mild, atypical attacks of rheumatism. These children are brought to the clinic for

vague symptoms such as pallor, listlessness, poor appetite, shortness of breath, and loss of weight. Often they run a slight temperature, suggestive of tuberculosis. Endocarditis may be the primary manifestations of the rheumatic cycle, arthritic manifestations and chorea appearing later on.

Again, a child may be brought to the physician on account of pain referred to the epigastric region, and the examination reveal a pericarditis. From these facts, the importance of routine examination of the heart becomes apparent.

A murmur alone is not sufficient evidence upon which to make the diagnosis of endocarditis. One must demonstrate the presence of enlargement of the heart as well. In fact, in acute myocarditis of diphtheria and pneumonia, and in the dilatation of whooping-cough, increase in the area of deep cardiac dulness, together with a systolic apical murmur, is frequently encountered. These physical signs, however, are only of temporary duration, and if the heart regains its normal vigor and tonus they disappear.

A murmur at the aortic area rarely occurs independently of mitral disease, and it is usually diastolic or double. An uncomplicated systolic murmur at the aorta is suggestive of congenital heart disease. (Still, *Common Disorders of Childhood*.)

Accidental murmurs are rare in infancy, but not uncommon in childhood. They occur either as a so-called functional murmur over the pulmonary area in acute febrile disturbances and anemic states, or at the apex at the end of inspiration. The name cardio-pulmonary murmur has been given to the latter condition; it is most frequently heard in a vigorous, overactive heart. A constant murmur found in a child under three years old, especially if it be heard most distinctly at the base, may be looked upon as congenital.

Still (*loc. cit.*) describes a murmur which he calls "physiological bruit," occasionally heard in young children, between two and six years old. It is usually heard just below the level

of the nipple, about midway between the left margin of the sternum and nipple line. In time it is systolic, and it is not transmitted. The character is twanging, somewhat musical, very like the noise made by twanging a piece of tense string.

Adherent pericardium is difficult to diagnose. Apert (*loc. cit.*) maintains that the only definite physical sign upon which a positive opinion can be based is the invariability of the situation of the heart in diverse bodily positions of the patient. This is demonstrated by noting the position of the apex beat and deep cardiac dulness with the patient standing, recumbent and lying on the right side. The chief clinical symptom in his belief is progressive asystole rebellious to any form of treatment. Personally I am of the opinion that systolic retraction of the chest wall in the cardiac region, if properly interpreted, is a diagnostic sign of great value, as is also a heaving, diffuse impulse associated with diastolic shock and diastolic collapse of the jugular veins. In a heart that is not markedly enlarged these signs are even of greater significance.

Treatment.—The treatment of heart disease resolves itself into prophylaxis, care during an acute attack or during an exacerbation of an old endocarditis, and the care of the child during periods of quiescence.

The chief prophylactic measure in the present state of our knowledge of rheumatic infection is attention to the throat. All colds in the head should receive prompt attention; adenoids and diseased or abnormal tonsils should be removed. Attacks of laryngitis and bronchitis, especially if recurring in nature, should be viewed with suspicion.

The diet should be generous and nourishing, and largely lacto-vegetarian, although meat is not contraindicated. Owing to the tendency to anemia, eggs and meat must enter into the diet with moderation. Fats are of the greatest importance, and in the winter time cod liver oil should be given.

Cold baths are not well tolerated by rheumatic subjects, and a damp, chilly climate is a disadvantage to these cases,

although clear, cold weather is beneficial. The clothing should be of wool throughout in cold weather. Exposure to others suffering with colds and sore throat is to be strictly avoided.

During an attack of endocarditis, absolute rest in bed must be enforced until the pulse and temperature have been normal for two or three weeks. There is less danger of the patient becoming weak from lack of exercise than there is of straining a weakened heart muscle, and relighting the endocarditis. General massage may be employed during the period of convalescence.

At the end of this period also, if the heart remains enlarged and shows deficient tonicity and the peripheral circulation is poor, the Nauheim baths and resisted movements may be employed to great advantage.

Aconite is the best remedy in the early stages, when the inflammatory process is at its height and the pulse is rapid and the patient nervous and restless. Chilliness, palpitation, stitches about the heart, precordial distress and anxiety are keynotes for this remedy.

Bryonia is pre-eminently indicated in pericarditis, but its specific affinity for serous membranes also makes it valuable in endocarditis. When the patient is weak and indifferent, with continued fever, headache, great thirst, general rheumatic aching and soreness in the cardiac region, *bryonia* is the remedy indicated. In endocarditis and pericarditis, with violent palpitation and stitching pains about the heart, *spigelia* is indicated.

In myocarditis *arsenicum alb.*, *cactus* and *digitalis* are to be considered. When decompensation occurs, we may have to depend upon the physiological effect of *digitalis* to tide the patient over the crisis. *Convallaria* is a remedy which should also be considered in this condition, especially when the pulse is rapid and irregular, and the patient is conscious of the disturbed heart's action.

After the acute manifestations have subsided, the *iodide of arsenic* 3x trit. administered for several weeks often improves the child's general condition and cardiac action markedly. *Fer-rum phos.* may also be of value if there is a secondary anemia with shortness of breath on exertion and *kali carb.* when there is a slight edema of the ankles at the close of the day.

For general rheumatic disturbances *rhus tox.* is most frequently indicated. The recurring rheumatic sore throat suggests *guiacum*. An occasional dose of *sulphur* may be administered where chronic arthritis or cutaneous manifestations are noted.

MYOCARDITIS.

Acute degenerative and inflammatory changes in the heart-muscle are of frequent occurrence in the acute infections of childhood. The toxins of diphtheria, scarlet fever and typhoid fever are especially concerned in the production of myocardial degeneration (Romberg). True inflammatory changes—myocarditis—are most frequently associated with endo- and pericarditis, and are due to the invasion of the heart-wall with pyogenic organisms, chiefly the streptococcus pyogenes, staphylococci and pneumococcus (Ziegler). Myocardial changes have also been observed in whooping-cough by Koplik and Osler. Pyrexia is a contributing cause, but does not seem able to produce myocarditis by itself.

The varieties of degeneration encountered are granular, hyaline and vacuolar. All of them may have more or less fatty changes associated. The process may be purely degenerative throughout, but, as a rule, exudation and cell proliferation in the connective tissue stroma is associated therewith. In infectious and pyemic cases areas of round cell infiltration play a prominent role, which may break down, resulting in small intramural abscesses.

At autopsy the heart is found of a pale, yellowish-brown turbid color and the muscle is easily torn. It is the soft-heart of the older writers. The process is mostly diffuse, although

in true myocarditis the changes may be more pronounced in different areas.

The *symptoms* are essentially those of a weak heart. When myocarditis develops during the course of typhoid fever or pneumonia the pulse becomes weak and often irregular, the pulse respiratory ratio is too high and the first sound of the heart too weak to be accounted for by the fever alone. In the absence of demonstrable peri- and endocarditis we suspect that we are dealing with a degenerated myocardium. In the course of diphtheria the child may suddenly be seized with epigastric pain, vomiting, syncope; rapid, irregular pulse. Such symptoms are of the gravest prognostic significance.

The softening of the heart muscle invites dilatation; there is, therefore, usually some dilatation, especially of the right ventricle. A faint apical systolic murmur may be present. The heart is usually rapid and embryocardiac in rhythm. Bradycardia may develop, especially after diphtheria. On the other hand, there may be no symptoms, or only a short time before death will there be sufficient indications to make us suspect myocarditis.

The *diagnosis* of myocarditis cannot always be made during life, but there are certain symptoms that strongly point to its existence. The subject is well summarized by Koplik (*Med. News*, March, 1900) as follows: Attacks of faintness, pallor, vomiting; disturbed and irregular heart's action; persistent distortion of the respiration and pulse ratio as in adherent pericardium. When these attacks show a tendency to recur they are certainly significant. Physical examination reveals a weak apex-beat, weakness of the first sound or loss of its muscular quality, greater intensity of the second sound at the apex and accentuation of the pulmonary second sound. In pertussis there is in addition slight systolic blow at the apex, edema of the face and extremities, pallor, cyanosis and drowsiness.

The *prognosis* is grave. Under long-continued rest the heart may regenerate sufficiently to resume its function as before,

providing the changes have not been too extensive. The symptoms described as indicative of myocarditis are in reality due to dilatation (Osler). The abrupt death in the course of an acute infectious disease results from cardiac paralysis.

The *treatment* calls for the most complete rest. As long as symptoms show the slightest tendency to recur the child should not be permitted to sit up or make the slightest physical exertion. The remedy most homeopathic to the degenerative changes is *phosphorus*, and it is undoubtedly of value. The *iodide of arsenic*, *cactus*, *digitalis* and *kali carb.* should also be studied in connection with the case (see treatment of "Endocarditis"). Mild alcoholic stimulation is usually of value. As an emergency remedy, Holt speaks highly of *morphia*.

CHAPTER XI.

DISEASES OF THE KIDNEYS AND URINARY TRACT.

Albuminuria occurring during the course of the acute infectious diseases is the most frequent renal disturbance encountered in childhood. It is usually due to an acute parenchymatous degeneration of the kidney and not to an actual nephritis and is, therefore, a transient condition. True nephritis is rarely seen excepting as a complication of scarlet fever. This form of nephritis is described as "post-scarlatinal nephritis."

Malformations of the kidney such as congenital hydronephrosis, congenital cystic kidney and the horse-shoe kidney which represents a fusion of both kidneys, are occasionally encountered in autopsies upon infants. Hydronephrosis may be unilateral or bilateral, presenting a fluctuating tumor in the flank. The cystic kidney may be unilateral and if the cysts are of large size a firm, smooth renal tumor may be palpated. Cystic kidney is often bilateral, the kidneys not being enlarged but presenting a surface studded with small cysts of varying size. There may be sufficient normal renal tissue present in these cases to be compatible with a fair degree of health and a renal lesion may, therefore, remain unsuspected.

Tumors of the kidney are usually malignant, being either sarcomata of the embryonal adenosarcoma type or hypernephromata. Hypernephroma originates in the supra-renal gland or from adrenal cells abnormally distributed in the renal tissue and is characterized by its rapid growth and tendency to metastasis. Sarcoma likewise is prone to induce metastasis in the bones and distant organs and for this reason renal tumors in childhood usually terminate fatally within a year even if the tumor is extirpated.

The symptoms are a rapidly growing, hard, rounded tumor of irregular shape first appearing in the loin and encroaching

upon the abdominal cavity. It is usually painless although it may become sensitive from localized peritonitis. Hematuria is frequently detected. With the increasing size of the tumor pressure symptoms appear and as metastases develop cachexia, cough and bony enlargements make their appearance.

ALBUMINURIA.

Albuminuria occurring during the course of one of the infectious diseases is ordinarily due to *acute parenchymatous degeneration* of the kidney and is, consequently, a transient disturbance. The pathological changes are confined to the tubules of the kidney and result from the presence of soluble toxins in the blood. Certain drugs as well as endogenous chemical toxins originating in the intestinal tract may cause the lesions of a "tubular nephritis" for which reason albuminuria is a common symptom of the acute intestinal disturbances of infancy as well as of the acute infectious diseases.

The symptoms of acute degeneration of the kidneys are a high colored, clear, concentrated, acid urine containing a trace of albumin and a few hyaline casts, renal cells and blood corpuscles. There is no edema or tendency toward the development of uremia. The only physiological disturbance noted is a delayed sodium chlorid and potassium iodide excretion. The albuminuria disappears with recovery from the primary disease of which it was but a clinical manifestation.

POSTURAL ALBUMINURIA; LORDOTIC ALBUMINURIA.

When albuminuria occurs in a young individual the point to be decided is whether we are dealing with a case of nephritis or whether the albuminuria is purely a functional and consequently a temporary disturbance. There is a form of albuminuria which is not uncommonly encountered at the time of adolescence which, if carefully studied, proves to be of functional origin and unless the true nature of the condition is recognized a serious error in prognosis may be made and inappropriate treatment instituted.

Richard Bright in 1827 established the fact that the presence of albumin in the urine is an indication of inflammatory changes in the kidney and that patients who were afflicted with an albuminuria showed the signs of a nephritis at autopsy or developed the characteristic clinical manifestations of renal disease. For many years after this important discovery was made it was believed that every case of albuminuria was a case of renal disease, or Bright's disease. Even now this attitude is maintained by many clinicians without searching for further evidence of renal pathology outside of the presence of albumin in the urine. At the present time, however, the careful clinician realizes that albuminuria does not invariably spell Bright's disease. Leube repeatedly discovered albumin in the urine of soldiers after physical exertion; the albumin disappeared after rest and was never present in the urine voided in the morning on rising. Pavy made the discovery that among children and young adults cases of albuminuria could be found in which the albumin was only present at intervals, or cycles, and he designated this condition as "cyclical albuminuria." Heubner later shows that the albumin appeared when the erect position was assumed and that it was the change from the the reclining to the erect position, particularly in the morning hours which was responsible for the albuminuria. The urine voided when first arising in the morning was free from albumin in his cases while that voided several hours after being up and about gave a strong albumin reaction. Realizing the importance of posture as an etiological factor he called the condition "orthostatic albuminuria."

The etiological relationship of posture to this form of albuminuria has been fully established by the observations of numerous clinicians. Jehle, however, insists that posture is not the important factor but that a lordosis, involving especially the upper lumbar vertebra, is the actual cause of the condition and he has coined the term "lordotic albuminuria" as a substitute for postural and orthostatic albuminuria. While the majority

of patients with an orthostatic albuminuria present a lordosis as well as other evidences of physical inferiority, such as the Stiller type of physique, nevertheless, we encounter many children and young adults with these physical defects who do not have an albuminuria.

The evidence of organic renal disease is lacking in these cases. There are no cardiovascular changes, in fact the blood pressure is abnormally low, especially the diastolic. There is no edema of the extremities, ocular disturbances nor is uremia to be feared. There are no casts in the urine and the albumin is part nuclealbumin. The prognosis is good. As the child's physical condition improves the albuminuria improves, in fact we are justified in promising the parents that the child will "grow out of the condition" in the literal sense of this phrase.

The treatment of orthostatic albuminuria must therefore be a building up process and a diet rich in milk, eggs, fats, an abundance of green vegetables and meat in moderate amounts is indicated. Fresh air in abundance, out-door exercise and corrective gymnastics if lordosis and enteroptosis be prominent symptoms are to be followed out. Remedies are not required for the albuminuria; casts are seldom found in the urine. Such constitutional remedies as *calcareo phosphorica*, *phosphorus*, and *arsenicum* are useful, and *iron* if the child is anemic.

EDEMA WITHOUT KIDNEY LESION.

In protracted cases of enteric disturbances it is not uncommon to find general edema without any clinical evidence of nephritis. There is puffiness of the eyelids and a cushion-like swelling on the dorsum of the hands and feet. General anasarca may develop. There is usually a history of an acute gastro-intestinal disturbance associated with prolonged feeding of barley-water, broths and weak milk mixtures. It is, therefore, most likely partly toxic and partly dietetic in origin. The edema promptly disappears as soon as the infant's general condition is improved by rational feeding.

HEMATURIA; HEMOGLOBINURIA.

Hematuria, or blood in the urine, has the same significance in infancy as in later life, although it is much less frequently due to organic and mechanical causes (papilloma, calculus) than to acute nephritis, tuberculous cystitis and general disturbances, such as hemorrhagic disease of the new-born, purpura, scurvy.

Hemoglobinuria, or hemoglobin in the urine, results from the action of some toxic agent or ferment upon the blood, through which the hemoglobin is dissolved out of the corpuscles and excreted with the urine.

It has been observed in various infections (malaria, scarlet fever), in helminthiasis, after exposure to cold as a result of certain drugs (*potassium chlorate*, *phosphorus*, *arsenic*). The most striking form is *recurring hemoglobinuria*. This usually affects children whose health is below the normal standard and in many cases there is evidence of hereditary syphilis.

ACUTE NEPHRITIS.

Two distinct types of acute nephritis are encountered in childhood, namely *acute parenchymatous*, or *tubular nephritis*, and *acute diffuse*, or *glomerulo-nephritis*. Both are due to bacterial toxins which reach the kidney through the circulatory blood and set up an inflammatory reaction in its parenchyma. Tubular nephritis may be induced by bacterial and other toxins and by certain drugs and is the variety usually encountered complicating the various acute infectious diseases. The most severe type of tubular nephritis is encountered in diphtheria. The most important clinical type of acute nephritis, however, encountered in childhood is the glomerulo-nephritis which follows scarlet fever. This is also known as post-scarlatinal nephritis and it differs both pathologically and clinically from the other forms of nephritis seen in childhood. Some cases of glomerulo-nephritis are attributed to tonsillitis and septic

(streptococcic) sore throat. In such cases there has probably been an unrecognized scarlet fever since the rash may have been overlooked although the throat symptoms were noted. In recent cases of nephritis it is not uncommon to find signs of desquamation still in evidence although the child was not known to have had scarlet fever.

Pathology.—The kidney may or may not show gross changes. Usually it is enlarged, congested and the capsule is tense but not adherent. The cut surface is grayish or of a mottled red appearance and the parenchyma is soft and mushy. Punctate hemorrhages are usually dispersed throughout the parenchyma and blood and serum ooze from the cut surface. The glomeruli are seen as red or grayish points in the glomerular type. The pyramids are of a dark red color and stand out in sharp contrast to the cortex.

Necrotic changes in the epithelial cells of the tubules with some inflammatory reaction of the interstitial tissue are the chief findings in tubular nephritis. In severe diphtheritic cases the desquamative changes are very pronounced. In glomerulonephritis all of the renal elements are involved so that the term acute diffuse nephritis may be used synonymously with glomerulonephritis. However, since the glomeruli are especially attacked by the scarlatinal virus and since the renal function is so seriously crippled by this lesion, the term glomerulonephritis is both a good clinical and pathological one.

Symptoms.—The presence of albumin in the urine and a few hyaline and granular casts in a patient with an acute infection is significant of an acute parenchymatous degeneration of the kidneys. With the development of an actual nephritis the urine becomes scanty and high colored; it contains a large amount of albumin together with red and white blood corpuscles, blood and epithelial casts and renal epithelium. The clinical manifestations of tubular nephritis are not characteristic excepting for the urinary findings mentioned. In glomerulonephritis, however, dropsy and uremic manifestations develop in conjunction with the albuminuria and cylindruria.

A primary nephritis is ushered in with high fever, pain in the region of the kidneys, headache and vomiting, scanty urine and later anasarca.

When secondary to an infectious fever the symptoms develop less abruptly. They make their appearance at the height of the fever or during convalescence as occurs in scarlatina. Frequently a renal affection is not suspected until dropsy and scanty urine are noted. Post-scarlatinal nephritis appears, as a rule, in the third or fourth week of the disease.

Dropsy is naturally most noticeable in those portions of the body possessed of loose areolar tissue, and for this reason the face, particularly the eyelids, the wrists and ankles, legs and scrotum, become most markedly affected. The pleural and peritoneal sacs may also become involved.

Dilatation of the heart, indicated by an increase in the area of cardiac dulness and weak pulse, is a frequent complication arising during the course of nephritis. The urine is diminished in quantity, the specific gravity high, although the amount of solids excreted is far below the normal. Its color is dark-red or smoky, the latter indicating the admixture of blood and it contains a large amount of serum albumin; leucocytes; renal epithelium and casts. Blood and narrow hyaline casts occur early; later epithelial, granular and broader hyaline casts make their appearance.

Dropsy is frequently the first symptom noticed. In other cases uremic manifestations, namely headache, vomiting, suppression of urine—are the first clinical evidence of the disease. In favorable cases the anasarca promptly subsides under treatment and the albumin and casts disappear from the urine in the course of a few weeks. The severe type of cases with suppression of urine and uremic symptoms frequently prove fatal. Instead of the coma and convulsions of uremia the air hunger (hypernea) of acidosis may develop and the child die with symptoms of acidosis in place of those of uremia.

The outlook as to the ultimate condition of the kidneys in

cases which have recovered from the acute symptoms is generally good. A trace of albumin may persist for months in the child's urine but few cases of chronic nephritis in adults can be traced back to an attack of acute nephritis in childhood. The possibility, however, of a latent nephritis persisting throughout adolescence should always be borne in mind.

Treatment.—Prophylactic measures should be carried out during the course of all acute infectious diseases, particularly during scarlet fever. The urinary output should be kept high by giving the child water freely and a low protein diet should be enforced. The chief foods allowed should therefore be fruit juices, milk and cereals. The child should be kept in bed for some time during convalescence especially if the renal function be impaired and the body surface carefully guarded against being chilled. Daily tepid sponge baths should be given to maintain a good skin function. Daily bowel movements should also be secured through the use of enemata.

When *anasarca* develops gentle sweating may be induced by means of warm packs but this procedure must not be carried to the extreme. The amount of fluids administered should be somewhat reduced but not entirely cut out. A mild saline laxative may be given daily in place of the enema.

For *uremic* symptoms and suppression of urine a warm bath may be given followed by a gentle sweat and a high, warm rectal enema administered. Salt should not be added to the enema as it interferes with the renal elimination. A teaspoonful of bicarbonate of soda to a quart of water, however, is advantageous as there is usually a certain degree of acidosis associated with the uremic symptoms.

Arsenicum is indicated by the anemia and *anasarca*, especially prominent about the eyelids in the morning. There is scanty urine, the characteristic thirst and restlessness, and cardiac involvement.

Apis is frequently called for, and is most useful for conditions which arise suddenly, especially during the course of an acute

disease; the urine becomes scanty or suppressed, general dropsy develops, and pulmonary edema may set in. Other characteristic symptoms of *apis* are cerebral involvement, with coma, shrill, piercing cry, and convulsions.

Cantharis is strictly homeopathic to acute nephritis and has been found a useful remedy by both schools of medicine. It is helpful for the very acute symptoms which may arise, such as high fever, tearing pains in the kidneys, vesical tenesmus, retention of urine and uremic coma; also in the later stages, to remove the albumin from the urine.

Hepar.—Urine decreased in quantity and containing blood, albumin and hyaline casts. Kafka's experience was "No remedy will act quicker or surer than *hepar sulph.* 3. in the case of dropsy and albuminuria occurring during scarlet fever" (*Homeopatische Therapie*). His reason for using this remedy was on the grounds of the relationship of *hepar* to croupous inflammation.

Lachesis and *terebinthina*, especially the latter in post-scarlatinal nephritis, are indicated in hemorrhagic nephritis. In *lachesis* the urine is very dark in color, and the characteristic subjective symptoms of the drug may be present. The urine indicating *terebinth*, is highly albuminous and scanty, the color being "smoky," due to the abundant admixture of blood. *Helleb.* is also prominent in hematuria.

Uremic convulsions call for *cicuta*, *bell.*, *hyos.* or *stramonium*; the *arsenite of copper* seems particularly applicable to all forms of uremic conditions and is the remedy most to be relied upon.

All complications, such as serous effusions, edema of the lung, etc., must be dealt with purely symptomatically. The resulting anemia most frequently calls for *arsen.*, *kali carb.*, *ferrum metallicum*.

CHRONIC NEPHRITIS; BRIGHT'S DISEASE.

Chronic nephritis may develop from an acute nephritis, especially after the post-scarlatinal form. It may occur as a complication of a chronic infectious disease such as tuberculosis, syphilis and long-standing suppurative processes. Often it is idiopathic, with perhaps a hereditary factor in its etiology. There are two types of chronic nephritis, the *parenchymatous* and the *interstitial*.

CHRONIC PARENCHYMATOUS NEPHRITIS.

In this form the kidney is much enlarged, presenting a yellowish-white appearance (large white kidney). On section, the cortex is found thickened and swollen and light in color, while the pyramids retain their dark-red hue.

The epithelium of the tubules is swollen and degenerated; the tubules contain degenerated cells and coagulated fibrin. Hyperplasia of the interstitial connective tissue and nuclear proliferation in the glomeruli and their capillaries together with amyloid degeneration of the smaller vessels, are the histological lesions. Delafield sums up the pathology of the whole condition in the name he gives it, namely, chronic productive nephritis with exudation. Amyloid changes in the blood vessels of the glomeruli are common in the nephritis of childhood.

Symptoms.—As a rule, the first symptom leading us to suspect a nephritic condition, aside from the discovery of albumin and casts in the urine, is anasarca. This is first noticed as a puffiness of the face, especially of the eyelids. Often there is a rapid increase in the dropsical manifestations and the legs share in the edematous process while the abdomen becomes more and more distended until an ascites can be detected.

The renal function is markedly disturbed. There is a marked reduction in the urinary output and the specific gravity is relatively low. There is consequently defective elimination of nitrogenous waste with an increase of non-protein nitrogen in

the blood. A relative acidosis also develops from faulty excretion of inorganic salts. The urine shows a high percentage of albumin and microscopic examination reveals degenerated epithelium; hyaline, granular, epithelial and, at times, fatty casts.

The *course* of the disease may cover a number of years with periods of remissions and apparent cure. Some cases undoubtedly do make a complete recovery for this is more likely to occur in a child than in an adult. In the majority of cases, however, the outlook is unfavorable, death ultimately resulting either from uremia or circulatory failure. The average duration is about two years.

Uremia is usually ushered in by headache and vomiting, followed by convulsions and coma. In children convulsions are more common than in adults; the same may be said of acidosis. Uremia is not so liable to develop when amyloid changes are marked in the kidneys, which can be suspected from the freer urinary secretion and the coexisting enlarged liver and diarrhea.

CHRONIC INTERSTITIAL NEPHRITIS.

This is a rare form of nephritis in children, and its etiology is unknown. Syphilis, tuberculosis, arteriosclerosis and heredity have been considered as causes, and in some instances it has apparently followed an acute infectious or eruptive fever. Allan Baines (*Archives of Pediatrics*, 1901) reports a pronounced case of arterio-sclerosis with interstitial nephritis occurring in a boy ten years old. The etiology in this case was obscure excepting that he had rheumatism and chorea. In the cases which I have personally seen no etiological factor could be determined. Guthrie has lately reported seven cases in the "Lancet." He considers it, not a product of parenchymatous atrophy, but an interstitial inflammatory process with round cell infiltration of the stroma of the kidney, beginning in the cortex and spreading in the form of bands to the center of the organ.

The *urine* is pale and abundant, low in specific gravity, and contains a small percentage of albumin, which may only be present at certain times. Such an albuminuria occurring several years after an infectious disease, the albumin being especially found in the morning urine, together with hyaline and granular casts, is a strong evidence of interstitial nephritis.

Dropsy seldom develops, but persistent gastro-intestinal symptoms and certain nervous disturbances, such as periodic headaches, vertigo, or convulsions, together with high arterial tension and hypertrophy of the heart, are indicative of contracted kidney, even in the absence of albumin. The *prognosis* depends much upon the compensation and integrity of the circulatory system, and the *course* is more protracted than in parenchymatous nephritis. Uremia or cerebral or other hemorrhage terminate the disease.

Treatment.—The diet should be restricted in nitrogenous food but the minimum protein requirement of the body must be met by a certain amount of protein food. This is about 1.5 gms. for every kilogram of body weight. When uremic symptoms develop all protein must be excluded from the diet. In the average case of nephritis milk is the ideal food; it should hold the most prominent place in the dietary, and it can be modified in many ways to vary the monotony of its administration. Fresh vegetables, fruit and cereals, are all allowable. There is danger of giving too much water and other fluids in these cases, as von Noorden has pointed out. The damage done to the heart and arteries may be greater than the good accomplished by this excessive “flushing of the kidneys.”

The function of the skin should be promoted and the cutaneous circulation stimulated by tepid sponge-baths, followed by vigorous rubbing. The undergarments must be of wool, to protect against any sudden chilling.

Water should be drank regularly between meals, in moderation; such springs as Poland, Bedford and Waukesha, or a distilled water, are especially beneficial in keeping up a sufficient excretion of urinary solids.

The measures recommended for dropsy and uremia under *acute nephritis* are equally applicable here. Diuretics are contraindicated in the edema of nephritis. The remedies most useful for the nephritis itself are *apis*, *arsenicum*, *aurum mur.*, *canth.*, *merc. cor.*, *phos.* and *plumbum*. These remedies are strictly homeopathic to the pathological process in the kidney, and have proven themselves of great clinical value. *Aurum* and *plumbum* are particularly related to the interstitial form of nephritis.

DIABETES INSIPIDUS.

Diabetes insipidus is a chronic form of polyuria occasionally encountered in childhood, but like diabetes mellitus it is a comparatively rare disease. It differs from diabetes mellitus in the absence of sugar in the urine and from the polyuria of interstitial nephritis in the absence of high blood pressure and absence of albumin or casts in the urine.

The *etiology* and *pathology* are obscure; heredity, traumatism to the nervous system and organic brain disease, however, seem to bear distinct relationship to some cases, and it has occasionally developed after the infectious fevers. Disease of the pituitary gland may be responsible for some cases.

The pathognomonic *symptoms* are polyuria and great thirst; the urine is pale and limpid, of low specific gravity, and contains neither sugar nor albumin. Associated symptoms are loss of weight, impaired digestion, constipation and functional nervous disturbances. The onset is usually gradual and the course a prolonged and tedious one, either ending in recovery or in death from exhaustion or some intercurrent affection. The *prognosis* is not altogether unfavorable, especially in the case of children, but a definite cure cannot be promised.

Differential diagnosis rests between *diabetes mellitus* and *interstitial nephritis*. From the former it is readily distinguished by the low specific gravity of the urine, the absence of sugar, as well as lack of marked and rapid emaciation. *Interstitial nephritis* is associated with arterio-sclerosis, high blood

pressure, hypertrophy of the heart and uremic manifestations and repeated careful examinations of the urine seldom fail to find albumin and hyaline casts. The ingestion of salt increases the urinary output in diabetes insipidus while in nephritis there is delayed salt excretion. *Hysterical polyuria* is emotional in origin and only a temporary disturbance.

The remedy which has yielded the most satisfactory results in my hands in cases of persistent polyuria, when the patient urinates freely every hour or two during the day, and from four to six times during the night, the urine being pale and limpid, is *natrum muriaticum*, 6th dilution. Thirst may be a prominent symptom, together with constipation, etc.

Ignatia is occasionally useful in highly nervous temperaments. Goodno has obtained positive results from *strophanthus* 1x. Hughes recommends *scilla* 2x; Schuessler, *ferrum phos.* 1x. The administration of pituitary gland promises to be helpful in some cases.

DIABETES MELLITUS.

Glycosuria is frequently encountered in young children during the course of an acute gastrointestinal disturbance and is one of the chief symptoms of Finkelstein's "Alimentary Intoxication." Such a glycosuria is, however, transient in character and does not indicate a permanent carbohydrate intolerance.

True diabetes is very rare during childhood, and its pathology, etiology and symptomatology are identical with the diabetes of adults. The course, however, is more rapid, and it is almost invariably fatal. The disease may terminate in a few months with diabetic coma; or if it is a mild case, amenable to treatment, it may run for years. The outlook for a case of diabetes in a child must necessarily always be unfavorable because the restricted diet which is necessary to control the diabetes proves inadequate for the needs of the growing organism and while the glycosuria may be held in check, serious malnutrition is the inevitable result of the treatment.

Diabetes is fortunately rare in children. The disease is being recognized more frequently and Joslin has found 4.7 per cent of his cases occurring during the first decade against 1 per cent of other writers on the subject.

The *etiology* is still obscure. Heredity no doubt plays an important factor. I have on several occasions encountered diabetes in the children of diabetic parents and have seen cases of two and three successive children in the same family succumb to the disease. Syphilis does not appear to be an etiological factor. Obesity is a predisposing cause in children as well as in adults; also overeating. Infections, no doubt, play an important role. Disease of the pancreas is the basal etiological factor. Cushing has called attention to the relationship of the hypophysis to glycosuria.

Symptoms.—Allen defines diabetes as deficiency of pancreatic amboceptor. According to his belief glucose normally exists in the blood in the form of a colloid, due to its combination with a substance which is probably the internal secretion of the pancreas and which he calls “pancreatic amboceptor.” When this substance is lacking the glucose acts as a crystalloid and thus produces diuresis and is also lost to the body tissues. Another important fact relating to diabetes is that the normal tolerance for carbohydrates is lost and that the more sugar is taken by the individual the more is lost and the lower his carbohydrate tolerance becomes. —(Allen’s paradoxical law.)

The pathognomonic symptoms of diabetes mellitus are polyuria, voracious appetite and great thirst, with usually constipation and indigestion, marked and rapid emaciation, dryness of the skin and nervous disturbances, such as formication and neuralgia. The urine contains glucose, and its specific gravity is high. Heightened susceptibility to infections, especially to tuberculosis is characteristic of diabetes. The greatest danger, however, lies in the development of diabetic coma, which usually causes the death of the patient. The coma is probably the culmination of a gradual increasing acidosis which has devel-

oped beyond the point of control by the various defensive mechanisms of the body.

Treatment.—The first step in the treatment is dietetic. Goodno recommends the employment of a diet absolutely free from carbohydrates until the glucose disappears from the urine, then gradually increasing the dietary and noting the effect of each new article upon the urine. Von Noorden's method of gauging the diet in diabetes is the most accurate and scientific of all recent contributions to the literature of this subject. It is clearly set forth by Lawrence in the *N. Amer. Jour. of Hom.*, Jan., 1904. When the acetone and diacetic acid are persistently present in the urine it is necessary to allow the patient a certain amount of carbohydrate. An occasional fast day, as carried out by Allen, usually clears the acetone from the urine and raises the sugar tolerance.

Meat, fish, eggs, all green vegetables of the 5 per cent carbohydrate class, fats and oils, gluten bread and butter-milk should constitute the diet list as far as possible. Water should be drunk freely.

Cases with acidosis which cannot be made sugar free by the ordinary diets may need an occasional continued fast until the urine contains neither sugar nor diacetic acid. In order to avoid the occurrence of coma in such cases the fast should not be abruptly started but fat should be omitted from the diet for a day or two; then continue a gram of carbohydrate per kilogram of body weight for twenty-four hours in the form of orange-juice or oatmeal gruel. Give water freely both by mouth and by rectum if necessary. Keep the patient in bed and warm during the fast. Move the bowels by enemata but avoid cathartics (Joslin). Bicarbonate of soda is helpful in acidosis but the excessive use of this salt has done more harm than good.

The following remedies have proven useful in diabetes both in respect to the carbohydrate intolerance and for the complications likely to occur.

Arsen.—Great emaciation and exhaustion; anemia; intense thirst; associated nephritis; complications, such as boils, gangrene, cutaneous eruptions.

Aurum mur.—Syphilitic dyscrasia; profound neurasthenia and mental depression.

Lactic acid.—Gastric disturbances predominate (*uranium nitr.*); dryness of tongue; empty feeling in epigastrium; constipation; stools hard and black; sluggish circulation in extremities. Administered in the lower dilutions.

Lyc.—This remedy is often indicated by its gastric symptoms together with the presence of uric acid in the urine.

Nux vom.—When the digestive tract is the main seat of disturbance; also neuropathic cases with many characteristic nervous phenomena, such as formication in the limbs; irritability; numbness and paretic condition of the lower extremities; gouty inheritance.

Nux, phosphoric acid and *arsenic* are perhaps the most frequently helpful remedies.

Phos. ac.—Cases of nervous origin. Profuse urination, with pain in back and region of kidneys, accompanied by great prostration, emaciation and sleeplessness. Rapid growing youths.

Uranium nitr.—Glycosuria. According to Prout, this remedy is especially useful when the disease originates in disturbances of the digestive tract, in contradistinction to *phosphoric acid*, which is indicated when it originates in the nervous system.

Rhus aromatica is a favorite remedy with the Eclectic school, who credit it with power to control the elimination of sugar through the urine. It is particularly indicated when there is incontinence of urine, being administered in doses of several drops of the tincture, three to four times daily.

PYELITIS.

Pyelitis, or inflammation of the pelvis of the kidney, is a comparatively common clinical condition encountered in infancy. It differs from the type of pyelitis encountered in adults in the fact that it is clinically a primary affection, while the pyelitis of the adult is usually a pyelonephritis secondary to stone in the kidney or is a manifestation of tuberculosis of the kidney or surgical kidney.

Infantile pyelitis is characterized by an acute onset associated with gastrointestinal symptoms, a febrile period of limited duration, the presence of pus and bacteria in the urine, the pus usually persisting for some time after the temperature has reached normal, and a strong tendency to relapses.

The etiological factor is in the majority of instances the colon bacillus. There is still a difference of opinion as to the mode of infection. For some time the belief that the bacillus reached the kidney pelvis by way of an ascending infection was the most popular one. This theory was based on the fact that pyelitis occurred most frequently in female infants and that during acute bowel disturbances the colon bacillus found its way readily into the urethra and could thence be carried readily into the bladder and into the ureters. Another mode of infection which was considered to be more probable from the anatomic standpoint is by way of the intestinal walls directly into the genitourinary tract. While such a mode of infection is very likely probable, still it does not seem likely to occur excepting in very rare instances. The most probable mode of infection in the majority of cases appears to be by way of the circulation. The grounds for this belief are that pyelitis is not a primary condition but develops during the course of an acute gastrointestinal infection. The child is sick with high fever, vomiting, distended abdomen and foul stools for several days before pus is discovered in the urine. After the pelvis of the kidney has become infected the urinary symptoms will persist

for some time even though the intestinal condition has been cleared up. Relapses also may occur either from a persisting renal focus or from re-infection from the intestines. The argument advanced against the theory of infection by way of the blood stream is that blood cultures in cases of pyelitis are negative. This argument, however, cannot hold for it is a well-known fact that bacteria may rapidly disappear from the blood in certain infectious diseases but may be demonstrated in the urine for a long period after the blood has become sterile. This is notably the case in typhoid fever in which disease blood cultures are only positive in the first week while the urine may still be strongly positive for typhoid bacilli in the third week.

Symptoms.—Pyelitis is rarely recognized from its symptoms since there is nothing distinctive about its clinical course. We should always suspect pyelitis as the most probable cause of an obscure fever in an infant if an infectious diarrhea or pneumonia can be ruled out. This applies particularly to the milder type of case in which fever is the chief symptom. The fever is continuous, usually lasts about ten days and has a distinct tendency to recur.

The more severe type of case is abrupt in onset with a distinct chill or its equivalent, high fever, vomiting, distended abdomen, offensive stools. Tenderness over the kidneys may develop and the child holds itself rigidly and shows evidence of pain in the back. Painful urination may also be observed. The clinical picture is at first confusing since the gastrointestinal symptoms are the predominating ones. There is often a history of some indiscretion in diet or a change of milk, and unquestionably the original disturbance is an intestinal one. The fever, however, continues in spite of catharsis and starvation diet and an examination of the urine at this time explains the reason for the persistence of the temperature.

The *urine* is diminished in quantity, acid reaction, cloudy, and on microscopic examination an abundance of pus and mobile bacilli will be found. A trace of albumin and a few

casts, indicating an associated toxic parenchymatous nephritis, may also be present. Under appropriate treatment the urinary output increases, it becomes pale and clear in color, the reaction becomes alkaline and pus gradually disappears.

Treatment.—In the milder types of pyelitis a careful supervision of the infant's diet, the use of a mild laxative such as milk of magnesia and the administration of several ounces of water between each feeding are the main indications for treatment. If the urine is rendered alkaline by the administration of bicarbonate of soda, the colon bacillus will find a less favorable environment for its existence and is more readily eliminated. An exclusive diet of butter-milk is useful in some of the more stubborn cases.

In the severe forms of pyelitis the intestinal tract should be thoroughly emptied by means of a dose of castor oil and the infant kept on barley-water, orange-juice and Mellin's food with water until starvation stools make their appearance. Butter-milk or albumin-milk may then be given. If the case is seen early, while the infection is still active and the urine acid in reaction, hexamethylenamine may prove useful. It may be given in doses of two grains every three hours. As soon as the urine becomes alkaline, however, this remedy should be discontinued as it is of no value under these conditions. The most important thing in the treatment of pyelitis is to urge the infant to drink an abundance of water. The water can be sweetened, if necessary, or flavored with fruit juices. Five grains of bicarbonate of soda may be added to three or four ounces of water and given every 2 to 3 hours. If the child cannot be made to take a sufficient amount of water by mouth, it may be administered by rectum, 4 to 6 ounces at a time. The best solution to use is one pint water, one level teaspoonful of salt, and one level teaspoonful of bicarbonate of soda.

The chief remedy for the acute symptoms of pyelitis is *cantharis* 3x dilution. *Aconite* is indicated at the time of the onset of the condition when chill, fever, and renal congestion

are the chief symptoms. If nervous symptoms predominate *belladonna* will be indicated. After pus makes its appearance in the urine and the pathological process in the kidney has become established *cantharis* should be given. *Merc. corr.* is indicated in the chronic type. *Autogenous vaccines* have proven useful in the recurring type to overcome the abnormal susceptibility of the renal pelvis to the colon bacillus.

ENURESIS.

A normal child acquires control over its bladder and bowels during the second year so that a child of two years will make its wants known and keeps itself clean. Control over the bladder during sleep is, however, a little later in its development, the average age at which a child is able to pass the night without wetting itself being about two and one-half years. Imbecile children never gain control over either the bladder or rectum while in the mentally and physically backward it is a late acquisition. Aside from the normal physiological development of the voluntary control of the function of micturition training is also a factor to be considered.

When an apparently normal child of three years or older still urinates involuntarily we speak of this condition as *enuresis*. Bed-wetting may persist up to the time of puberty or even later. Various sources of reflex irritation and physical defects have been blamed for the enuresis. Phimosis, adherent clitoris, adenoids and worms have occupied a prominent position in its etiology. Rarely, however, are the confirmed cases of enuresis helped to any extent by the removal of the conditions named, and they, therefore, cannot occupy a place of much importance in the etiology.

Abnormalities of the urine have also been looked upon as causative factors. Acid urine, uric acid crystals and oxalate of lime crystals in the urine, pyelitis and cystitis may occasionally be encountered in enuresis and aggravate the same but the urine rarely gives evidence of any of these conditions in the

great majority of cases. A polyuria undoubtedly exists but this is of purely nervous origin.

Enuresis is primarily a neurosis. The mechanism of urinating has remained so fixedly a purely reflex act that the bladder contracts and empties itself entirely independently of the will or consciousness of the child as soon as an afferent impulse of sufficient degree reaches the bladder centre in the lumbar cord. A normal child has learned to control the bladder by having gradually developed an inhibition over the involuntary discharge of urine. It becomes conscious of the sensation of a full bladder and the desire to urinate and through the inhibitory power of the higher cerebral centres over the involuntary centres in the cord is able to hold this desire in check. At the will of the individual the inhibition is relaxed and through the added help of the abdominal muscles the emptying of the bladder becomes a purely voluntary process.

The child afflicted with enuresis is very often unconscious of the process of urinating and may wet itself during the day, especially at play or when deeply interested in whatever is occupying its mind. The deep sleep natural to children is also looked upon as an etiological factor not permitting the desire to urinate to enter into its consciousness. Abnormal irritability of the spinal centre of the bladder is however, the chief cause of the trouble. The child with enuresis is, therefore, usually a neurotic individual lacking in stability and self-control; often afflicted with other nervous or psychic disturbances such as stammering, tics, somnambulism, night terrors, etc. There is often a neurotic family history or other children in the same family have enuresis. Anemia and malnutrition are often present but are not necessary factors. The child may appear to be normal mentally but it is usually found lacking in the ability to concentrate or apply itself for any length of time to any one particular pursuit. Since training is such an important element in the treatment, this lack of mental discipline offers one of the chief obstacles to success. Another difficulty in

eradicating enuresis when the condition is of long standing is the fact that it has become a habit. It is, therefore, of the greatest importance to institute systematic treatment before the process of micturition has become so fixedly a reflex act that the voluntary control over the bladder can only be acquired by the most painstaking training.

The *diagnosis* of enuresis should not be carelessly made. Nocturnal enuresis of irregular occurrence in older children should suggest the possibility of nocturnal epilepsy. Lack of control over the bladder present both day and night may be a symptom of a spinal lesion. The polyuria of *diabetes* is at times mistaken for enuresis and the urine of all cases should be examined as a matter of routine for sugar or evidences of a renal or genito-urinary affection.

Treatment.—In the majority of cases the child wets the bed during the early hours of sleep, that is, usually before midnight and the habit may become so firmly established that this occurs at practically the same hour every night. For this reason the child should not be permitted to have any liquids with its supper and it should be regularly taken up and made to urinate before it has had time to wet the bed. An important point in this connection is to entirely awaken the child so that it will be conscious of urinating. Many of these patients are such heavy sleepers that they can be taken up, made to urinate, and not be conscious of the event. The result is that they have the usual accident shortly afterwards just as if they had not been taken up.

Punishment is of no avail. However, suggestion, and training, are of the greatest help in educating the child to gain voluntary control over its bladder function. The object of training is both to strengthen the sphincter of the bladder and to increase the inhibitory voluntary control over the same. Both night and morning, when the child has a moderately full bladder, it should be taught to urinate at command and interrupt the process two or three times before the bladder is emptied. The mother should give the command to “start” and “stop” and the child soon learns to follow these instructions.

Sleeping in a cool, well-ventilated bedroom without too much bed clothing; cold sponge baths in the morning; the removal of all sources of reflex irritation such as phimosis, worms and adenoids and constitutional treatment for anemia or a neurotic temperament, are important and self-evident adjuvants. As to medicinal treatment, there is but one drug which possesses any specific value in enuresis and that is *belladonna*. It acts directly upon the nervous mechanism of the bladder and its effect may be compared to that of *digitalis* in auricular fibrillation. By blocking out the abnormal stimuli to the detrusor centre it lessens the irritability of the bladder and makes the acquisition of voluntary control possible. In this manner it helps to break up the habit of enuresis and is a most important element in the treatment especially in the beginning of a case until the results from training and general treatment can make themselves felt. The dose usually necessary is from five to ten drops of the tincture at bedtime.

VULVOVAGINITIS; GONORRHEA.

Vulvovaginitis is a catharrhal inflammation of the mucous membrane of the vulva and adjacent parts and in the cases encountered in hospital and dispensary practice is most frequently gonorrheal in origin. It is, therefore, always important to make a microscopic examination of a smear of pus from all cases of purulent vulvovaginitis. Koplik (1893) cultivated the organism, definitely proving its identity. This has been done since repeatedly.

Nonspecific vulvovaginitis is a simple catharrhal process due to lack of cleanliness; local irritation, such as smegma, seat-worms or masturbation; or it may be but part of a general catarrhal condition in scrofulous children.

There is a form of purulent vulvovaginitis and urethritis affecting both male and female children which is distinctly contagious and is due to a diplococcus which does not show, however, the staining and cultured peculiarities of the gonococcus (Koplik).

The *spread* of vulvovaginitis is surprisingly sure and rapid when children are brought into close contact, as in a hospital, for example. Every precaution should be taken, therefore, to prevent contagion.

Many children contract the disease from mother or nurse. It is often difficult to find the original source of infection. Rarer modes of transmission are by rape and attempted sexual intercourse.

The gravest aspect of gonorrhea is its *complications*. Salpingitis and peritonitis have been observed (Marx; Sanger). This leads to death or sterility. It is a rare complication. Infection of the eyes—ophthalmia—is constantly to be dreaded.

Arthritis in children is not infrequently gonorrheal. Holt and Kerley have observed that the majority of arthritides that were formerly looked upon as being septic are gonorrheal. Kimball (*N. Y. Med. Record*, Nov. 20, 1903) reports eight cases of pyemia with joint involvements in infants in all of which the gonococcus was demonstrated. No primary local lesion was present. The majority died during the height of the attack.

One of my cases, an infant three weeks old, developed ophthalmia three days after birth and a week later successive involvement of the shoulder, elbow and hip-joints. On one hand the second finger became involved in a fusiform swelling (dactylitis) and the wrist was also swollen. The temperature was continuously elevated, ranging from 101° to 102.5° F. Dr. Sappington obtained pus from the joints in which he demonstrated gonococci, verified by cultures on ascitic fluid and agar.

Treatment.—In the acute stage the local condition is much benefited by irrigation with a warm solution of a non-irritating silver preparation. One pint of a 1 to 500 solution of *protargol* or *albargin* may be used twice daily. The vulva and vagina are most satisfactorily irrigated through a small, soft rubber catheter, which may be gradually introduced into the vagina as the secretion from the external parts is flushed away. The vulva is then dried and dusted over with *boric acid*.

In the subacute and chronic stage *permanganate of potash*, 1-1000, may be used every day or two in a similar manner, followed by the dusting powder. Sometimes the dry treatment will give better results than douches.

In the early stages *cannabis indica* is indicated, or *cantharis* when there is dysuria. When the discharge becomes profuse and yellow *pulsatilla* is the most useful remedy. In chronic cases, *sulphur* or *sepia*.

CHAPTER XII.

DISEASES OF THE SKIN.

The skin of the newborn is exceedingly delicate and vulnerable and inflammatory reaction in the same is characterized by its intensity. It is very susceptible to infections and mild infections which in an older child induce only superficial pustule formation (impetigo) may be the cause of a form of pemphigus. The skin manifestations of congenital syphilis and of the exudative diathesis frequently make their appearance in earliest infancy.

At birth the entire body is covered with a waxy secretion, the vernix caseosa, which has served as a protective layer to the skin during intra-uterine life. The color is a deep red, owing to the vigorous surface circulation, and this condition persists for about a month. Usually desquamation of the epidermis, visible on close inspection, takes place during the second week. Jaundice, occurring on the third or fourth day, is encountered in about 80 per cent of all newborn infants.

The skin of the infant, as well as that of the newborn, is exceedingly tender and responsive to all forms of local irritation and infections. Functional disturbances also play an important role in the etiology of the skin diseases of childhood. Sweating is rare in infants, normally not noticed before the fourth month, and when persistent it is a strong presumptive sign of rickets. The use of irritating soaps, excessive bathing, and, on the other hand, uncleanness, are important etiological factors in the skin diseases of childhood, next to which improper feeding ranks. Dietetic errors no doubt are often directly responsible for the heightened irritability of the skin to factors acting from without as in the case of exudative diathesis. Slight mechanical and chemical irritation of the skin of such an infant is followed by a severe inflammatory reaction (intertrigo and

eczema). In some instances the excessive feeding of carbohydrate renders the child especially liable to pyogenic infections of the skin. Exposure to contagion, both to the parasitic skin diseases and to the acute exanthematous diseases to which children are so liable after they intermingle at school, is another reason for the frequent occurrence of skin affections and "rashes" among children.

Most of the skin diseases encountered in adult life may be seen in children. Only those conditions which are distinctive of childhood and which present certain characteristics distinguishing them from the type of the disease as seen in the adult will be discussed.

MILIARIA; PRICKLY HEAT.

Miliaria is a common form of rash seen in young infants and results from hot weather or too much clothing. It is due to excessive perspiration which causes a mild inflammatory reaction about the mouths of the sweat ducts. The rash consists of very superficial, closely aggregated, tiny red papules often interspersed with sudamina due to the occlusion of the mouth of a sweat gland. It is of sudden onset and under proper management fades out in a few days. The site of the rash is usually the chest and back, about the neck, and on the face. Itching is associated. There is no fever, but a febrile condition may be the cause of an attack of miliaria and so the condition is frequently mistaken for one of the eruptive fevers. Rachitic infants are especially liable to develop miliaria.

The *treatment* demands first of all the proper dressing of the child, removing excessive clothing and woolen undershirts where these have been worn and the correction of any underlying condition which is acting as a predisposing cause (rickets; intestinal indigestion). Sponging the body with a two per cent solution of boric acid followed by the generous use of a dusting powder gives prompt relief.

ECZEMA.

Eczema is an inflammatory reaction of the skin resulting from some form of local irritation and is indicative of a constitutional irritability of the skin rendering the individual susceptible to this affection. Pathologically it is a catarrhal type of inflammation which begins in the corium in discrete punctate areas whence the process extends to the epidermis with the development of erythema, papules and later vesicles upon the cutaneous surface. It develops in patches composed of finely aggregated papules which increase peripherally and may thus affect extensive areas. The lesions present indicate the stage of the disease. They are as follows, appearing in the order named:

The stage of *erythema* in which the skin is red, tense and slightly edematous.

The stage of *papules*, or *papular eczema* characterized by the appearance of small, red, closely aggregated papules forming plaques, or patches of eczema.

The stage of *vesiculation*, or *vesicular eczema* in which the papules are converted into fine vesicles filled with serous exudate.

The stage of *secondary infection*, or *pustular eczema*. Pustules are formed and when these burst or are broken by scratching the pus and serum which has collected on the skin surface dries into a crust. As the inflammation subsides and the oozing subsides the final *stage of desquamation*, or *eczema squamosum* develops. The various stages of eczema can usually be observed in different parts of the body as a new patch may arise in one locality while an older one is approaching the healing stage.

Infants who develop eczema usually show distinct evidence of the exudative diathesis. It may also result from overfeeding in breast-fed and is not confined to artificially fed infants. Heredity may be a factor as well as disturbances in the endocrine glandular system. Concerning the latter nothing definite is

known excepting that some types of eczema are benefited by the administration of glandular products. Protein sensitization is also noted in some cases and a definite relationship of the skin eruption with the feeding of certain proteins (eggs, wheat, nuts, etc.), can be demonstrated in these cases.

Two distinct clinical types of eczema are encountered in infants, namely the acute, moist type confined mainly to the head and face which occurs in fat, overnourished infants and the chronic disseminated type seen in undernourished infants.

The moist, crust-forming type of eczema (*eczema rubrum*) which is usually encountered in well-nourished infants first appears on the face and its onset should be suspected from the abnormal redness of the cheeks which is the starting-point of the condition. The eruption goes through the various stages of an eczematous inflammation in rapid succession, namely papules, vesicles, pustules, oozing and crust-formation. In typical cases the process spreads to the forehead, eyes and neck while the scalp is covered with a scurfy seborrheic dermatitis. Secondary infection of the scalp with the formation of boils frequently occurs and the superficial lymphnodes of the scalp and neck are enlarged. The eczema may extend to the body although in the majority of instances the head alone is affected.

There is marked itching as a result of which the child constantly scratches and rubs the affected parts thus keeping them in a state of irritation and favoring infection and spread of the process. Bleeding usually results from the relentless scratching of the raw, oozing surface. One of the chief difficulties encountered in the treatment of these cases is the relief of the itching and the control over the scratching.

Eczema rubrum usually develops during the first half of the infant's life and runs a stubborn course, marked by ameliorations and exacerbations. It responds more promptly to constitutional and dietetic treatment than the disseminated type and can usually be gotten under control in the course of several weeks and eradicated before the child is a year old.

The *chronic disseminated form* develops later in infancy, usually toward the end of the first year and may persist in showing some evidence of its activity for a number of years. It occurs in infants showing signs of malnutrition and the lesions are mainly seen upon the trunk and extremities. They consist of numerous disseminated areas of scaly patches. The bends of the elbows and of the knee-joints are localities where the disease is likely to be most persistent and constantly noted. There is less itching than in the moist form but it is very stubborn in its course and difficult to eradicate. The skin covering the buttocks is often infiltrated and thickened and kept in a constant stage of irritation from the urine and stools.

The **treatment** of eczema is both local and constitutional. In cases of eczema rubrum the amount of food should be cut down and the use of skimmed milk or buttermilk often results in immediate improvement in the symptoms. In the dry form of eczema the exclusion of carbohydrates from the diet is often beneficial. Cod liver oil is beneficial in both forms. It is always expedient to wean the infant from an exclusive milk diet as early as possible and add vegetable broths or strained vegetables to the diet.

Local measures should be mainly sedative in character. Bathing should be avoided and the skin should be cleansed with olive oil or other bland washes (starch-water; bran-baths; weak alcoholic solutions, 30 per cent containing one half of one per cent of salicylic acid). Itching may be controlled by means of olive oil and lime-water, equal parts, a one per cent solution of carbolic acid or a calamine lotion. A weak tar preparation, 10 to 20 minims of oil of cade to the ounce of vaseline is useful for the chronic dry form of eczema. In the acute stage of facial eczema a calamine lotion is the best application (zinc oxide, 4 drachms, powdered calamine and boric acid, a drachm of each; glycerine, one half to one drachm; three ounces each of rose-water and lime-water. When there is much itching ten to fifteen minims of carbolic acid may be added). An ointment

containing twenty grains each of calamine, zinc oxide and boric acid may be used in the subsiding stage of the inflammation. In order to get the best results from an ointment it is sometimes necessary to apply the same on pieces of soft linen and bind them to the affected part with a bandage or a mask. Scratching must be inhibited by fastening the arms with appropriate restraining measures. Dusting powders are most useful in cases in which the trunk and buttocks are affected, and in vesicular eczema. A satisfactory formula is equal parts of zinc oxide, boric acid and starch.

Belladonna is an excellent remedy for the acute hyperemic stage of eczema, promptly relieving the congestion and itching. In vesicular eczema *rhus tox.* is the most useful remedy. When pustules form *hepar sulph.* is indicated. Constitutional remedies like *calcareo carb.*, *graphites* and *sulphur* should be studied in conjunction with the case. *Thyroid extract* in small doses has frequently proven beneficial in the chronic type of eczema.

ERYTHEMA SCARLATINOIDES.

The clinical importance of this form of erythema rests upon its superficial resemblance to scarlet fever. Although the majority of cases are mild and evanescent in character, still there are such in which the entire body is covered with a rash in association with fever and constitutional symptoms.

The *etiology* is evidently a toxemia which may be of intestinal origin or due to idiosyncrasy to certain foods. Eating certain food (shell-fish) or tainted meat may give rise to an attack. It is sometimes associated with certain infectious diseases and with sepsis. Certain drugs, notably mercury, the salicylates, quinine and belladonna may produce a similar eruption. One of the characteristics of erythema scarlatinoides is its tendency to recur.

The *eruption* usually appears suddenly, although it may be preceded by headache, malaise and fever. The lesions are mostly confined to the face, neck, trunk and extremities. The

rash is usually composed of punctate macules which coalesce and give the skin a uniform scarlet color or it may be diffuse in character. When the rash is confined to the trunk and extremities it is very difficult to distinguish from scarlet fever. Usually, however, the eruption quickly fades, leaving none of the grave symptoms attending scarlet fever. Burning and itching may be annoying symptoms. Desquamation is marked in most cases and recurrences are common.

The differentiation from *scarlatina* rests upon the absence of exposure to contagion; the less general distribution of the rash and absence of strawberry tongue, sore throat and adenopathy; absence of albuminuria. Desquamation occurs earlier and does not persist as long as in scarlet fever.

FURUNCULOSIS; BOILS.

A *furunculus*, or *boil*, is an acute, deep-seated, circumscribed inflammation originating in a hair follicle or sebaceous gland and terminating in necrosis of these structures and surrounding connective tissue. The *cause* is infection with the staphylococcus pyogenes aureus.

Symptoms.—In certain infants with malnutrition, boils may become a very serious complication. They develop in great numbers, especially on the head. They are found chiefly on the scalp but may also develop on the face, shoulders and buttocks. The majority of furuncles are small but in some instances fairly large abscesses develop. While the staphylococcus is the exciting cause, still it appears that there is a predisposition in these infants which makes them such a favorable soil for this organism. Overfeeding with sugar has been mentioned as a cause as well as a latent tubercular infection.

In older children, furunculosis frequently accompanies or follows an attack of scabies or pediculosis. This will be readily understood by recognizing how irritated and inflamed the skin becomes as a result of the incessant scratching accompanying these parasitic diseases, thus inviting the entrance of pyogenic

organisms. Improper and tight clothing, irritating soaps, poultices, and the too lavish use of strong antiseptic lotions may be contributing factors.

Treatment.—Constitutional as well as local treatment is necessary in these cases. If there has been an excess of sugar in the diet this should be reduced and cod liver oil should be given. Orange juice should also be introduced into the diet.

Hepar sulph. is the best remedy not only for this condition, but also for all pustular dermatoses. An autogenous vaccine may be resorted to in stubborn cases resisting the usual forms of treatment.

Local treatment consists in incising the lesions as soon as pus has formed and washing the affected area with a weak solution (1 to 4000) of bichlorid of mercury to prevent spreading the infection. A dressing of 1 per cent Lysol may then be applied. As soon as a new lesion is discovered it should be touched with tincture of iodine with the hope of aborting the same.

IMPETIGO CONTAGIOSA.

Impetigo contagiosa is an acute contagious dermatitis, characterized by the formation of superficial, circular or oval vesico-pustules or blebs, which rapidly form yellowish crusts. The exciting cause is the *staphylococcus aureus*.

Symptoms.—Except in isolated cases, occurring in infants, no constitutional symptoms precede or accompany an attack. When present, however, they give rise to submaxillary and pre-aural adenopathy, together with moderate fever. The lesions are usually seen upon the face and hands. Where the fingers become involved the lesions are situated about the tissues surrounding the nails. Exceptionally, lesions are found on the trunks and extremities.

The lesions at first are minute vesicles, later increasing in diameter, becoming vesico-pustules. Their contents are sero-purulent. Desiccation rapidly occurs, leaving brownish spots,

which soon disappear. The attack lasts about a week, fresh crops appearing daily. Occasionally lesions rupture and coalesce, giving a honeycomb appearance to the group; under such conditions itching is a prominent feature.

In young infants and in the newborn impetigo, instead of manifesting itself as a pustular affection, presents the clinical manifestations of pemphigus. This disease, occurring in the newborn, is, therefore, known as *pemphigus neonatorum*, or *impetigo contagiosa bullosa*. It must not, however, be confused with sepsis of the newborn in which severe constitutional symptoms accompany the skin lesions.

Diagnosis.—Impetigo contagiosa may be mistaken for impetigo simplex, varicella, the pustular type of eczema, ecthyma, and pemphigus.

Varicella is differentiated by the presence of fine, pearl-like vesicles which appear in crops, and which in some instances leave cicatrices. *Varicella* is occasionally accompanied with grave constitutional symptoms. *Pustular eczema* may suggest impetigo simplex, although an eczema invariably produces more infiltration and more subjective symptoms. In eczema the lesions, although pustular, are deeper, and surrounded by an inflammatory areola. The lesions are found upon the legs, regions rarely attacked in impetigo contagiosa. *Ecthyma* is a disease of adult life.

Pemphigus is rarely met with in infants and children. The lesions are blebs. Constitutional symptoms are present. The resemblance of impetigo in the newborn to pemphigus is only superficial. In pemphigus the bullae are larger, are distended with fluid and appear in crops.

Treatment.—Warm baths should be given morning and evening. Crusts, if adherent, may be removed by soaking with olive oil. A mild antiseptic local application such as the ammoniated mercury, ten grains to the ounce of vaseline, will cure the majority of cases promptly. In young infants a calamine lotion containing one-half of one per cent carbolic acid

is preferable to an ointment. *Hepar sulph.* may be required to help eradicate the condition.

URTICARIA; HIVES.

Urticaria is an inflammatory cutaneous affection characterized by the appearance of evanescent pinkish elevations (wheals) which are accompanied by itching and other sensory disturbances.

Etiology.—Hives arise from causes that are both internal and external. Certain seasons are, in a measure, responsible for their outbreak; they are especially apt to appear in the spring and fall. The majority of cases occurring in children may be traced directly to some gastro-intestinal derangement. The condition is most likely one of idiosyncrasy to some food protein. Constipation, diarrhea, worms, and acute or chronic indigestion may occasionally be responsible. Improper clothing, low or high temperature, and the bites or sting of insects may be exciting causes.

Symptoms.—The lesion of urticaria is a wheal. This begins as a red, slightly elevated spot which enlarges, the centre becoming paler in color. In shape it is round or oval, frequently changing its size and locality, appearing from time to time upon different portions of the body. The lesions are particularly evanescent; they may last a few hours or but a few minutes, leaving behind no trace of their former presence. Rarely they persist for days; occasionally they coalesce and attain considerable dimensions. Their favorite seats are the extremities and buttocks, although they may appear on any portion of the skin or mucous membrane. Their outbreak is invariably attended with intolerable burning and itching, and a slight degree of fever. An attack may be acute or chronic. The acute attack is usually attended with gastric derangement, headache and slight fever. The eruption appears and disappears quickly, leaving no trace save a few scratch-marks, resulting from the itching. The chronic type may last for weeks or months. In young children

papules and vesicular lesions are frequently associated with the wheals. The latter are of an evanescent character but the former lesions are more persistent and so they may not be suspected of being due to the same etiological factor. The papular form is called *lichen urticatus* and is most frequently seen on the arms and legs. Another form seen in children is *urticaria pigmentosa* in which a pigmented spot persists after the disappearance of the wheals.

Urticaria papulosa, or *lichen urticatus* is a skin affection very common in childhood. It is characterized by the appearance of small, discrete, round papules—often beginning as a wheal, but persisting as an itchy eruption. A small vesicle often surmounts the papule. They are mostly confined to the extremities.

Diagnosis.—The character of the wheals, their evanescence and the presence of intolerable itching and tingling, are sufficient to establish the diagnosis. Dermatographism is also usually present and irritation of the skin, such as scratching, as a rule results in the appearance of a wheal at the site irritated.

Treatment.—Diet is of first importance in the treatment of urticaria. When directly traceable to a certain kind of food this article should be excluded from the diet. In some instances we may be able to determine the offending food by means of the cutaneous tests with the various food proteins especially prepared for this purpose. Constipation or diarrhea, if present, must be corrected. During an acute attack a saline purgative should be administered. Locally it may become necessary to allay the itching by applying a weak solution of *carbolic acid*, one-half of a drachm to eight ounces of water, or hot water to which has been added a little vinegar.

Belladonna may be administered early in the attack for the acute symptoms.

Urtica urens is indicated when itching, burning and tingling are prominent symptoms. It is indeed almost a specific. *Apis* is also frequently prescribed.

In the chronic form *hepar sulph.* 3x trit. has given me excellent results in a number of cases.

VEGETABLE PARASITIC DISEASES; TINEA.

The term *tinea trichophytina* is employed to designate a group of skin diseases due to a fungus growth. The body surface or the hairy scalp may be the seat of the lesions. Owing to the circular outline assumed by the patches of involved skin, tinea is commonly known as *ring-worm*.

TINEA TONSURANS; RINGWORM OF THE SCALP.

Tinea tonsurans is a highly contagious vegetable parasitic disease of the scalp, characterized by the presence of one or several bald spots, covered with scales and containing short broken-off hairs.

Symptoms.—Following a period of incubation, variously estimated at from three to five days, erythematous areas about the size of a twenty-five-cent-piece appear. They are covered with grayish scales, and are accompanied by slight itching; they enlarge peripherally and may coalesce. The hairs of these parts become lustreless and break off. In some cases the scalp is entirely denuded, making a complete bald spot. Occasionally vesicles and pustules form, and a certain amount of suppuration results. Resolution may take place in one area, while the disease is active in another. The general health is rarely affected.

Tinea tonsurans is due to the presence and growth of the trichophyton fungus. It is highly contagious, being transmissible to the lower animals, from whom it may be contracted. It is often endemic in asylums and hospitals, or where a number of children are congregated.

Pathology.—As a rule, only the superficial parts of the epidermis and hair are attacked in children. Microscopically, mycelia and spores are seen. The hairs become brittle, but, as

a rule, baldness is not permanent. The hairs usually return to their normal state.

Diagnosis.—Ring worm of the scalp may be mistaken for *alopecia areata* and *squamous eczema*.

Alopecia areata.—Baldness in *alopecia areata* is complete. The condition develops quickly and is more common in adults than in children. The patches are entirely devoid of hair and the affected skin has a white, atrophic appearance.

Prognosis.—Isolated cases, if seen early and subjected to proper treatment, are curable within a few weeks. An epidemic occurring where a number of children dwell together is hard to eradicate. In the majority of cases a few months will be required to efface the disease, and it must be remembered that relapses are common.

Treatment.—Prophylaxis is of the greatest importance. Children afflicted with *tinea tonsurans* should not be permitted to come in close contact with others. The possibility of contracting the affliction from domestic animals should be borne in mind. The general health of the child should be looked after and a constitutional remedy should be administered when there is anemia or malnutrition. When secondary infection with suppuration has set in *hepar sulph.* is indicated. Parasitocides are necessary to eradicate the fungus growth. Before these can be satisfactorily applied the scalp must first be suitably prepared. The hair about the patch and for some space surrounding it, should be cut and the scalp closely shaven. The short hairs should be removed by means of forceps. Scales and crusts, if present, are removed by scrubbing vigorously with a solution of green soap. Where the patches are extensive, it is necessary to shave the entire scalp. Depilation of the diseased hairs is tedious and often unsatisfactory. As a rule the hair is brittle and breaks off, not coming out entirely. The process, however, should be practiced daily. Locally the best application is *bichloride of mercury*, one to one thousand; it should be discontinued if it excites active inflammation. *Carbolic acid*,

one drachm to one pint of water, is frequently efficacious. Among other agents are *sulphur ointment*, a five per cent ointment of the *oleate of mercury*, and equal parts of the *oil of cade* and *olive oil*. After an apparent cure, the scalp should be treated every other day, to prevent the possibility of a relapse.

TINEA CIRCINATA; RINGWORM.

Tinea circinata is a highly contagious vegetable parasitic disease of the skin, caused by the *trichophyton* fungus. It is characterized by the presence of annular patches of varying size and character, occurring upon any part of the body surface. Ringworm is more common in children than in adults probably because the child's skin is more readily attacked by the fungus and because the possibilities for infection are greater. The fungus may be contracted from another child or from a domestic pet, most commonly the cat.

Symptoms.—Ringworm of the scalp and ringworm of the body are often found co-existing. Minute, irregular-shaped spots of a reddish-brown color indicate the commencement of ringworm of the body. Later a distinct circular patch is seen, which heals in the center and spreads peripherally. Around the margin of each patch small papules and papulo-vesicles are seen. Scaling is a distinct feature. The typical ringworm is usually about the size of a dime, and it stands out prominently from the surrounding skin. In some instances the rings join together. Any part of the body may become affected, although the face and hands are most frequently attacked. Next to these localities, the axillary and inguinal folds are involved.

Diagnosis.—*Tinea circinata* may be mistaken for *seborrhea* and *eczema squamosum*. In *seborrhea* the scales are greasy and the fungus is absent. In *eczema* there is infiltration of the skin, more itching and the characteristic annular formation of the lesion is absent. Should any doubt exist as to the diagnosis, a microscopical examination will usually detect the fungus.

Treatment.—The fungus can be destroyed by scrubbing the

lesions every morning and evening with green soap and hot water, and afterwards applying a solution of *sodium hyposulphite* (drachm to the ounce) or painting the patch with a weak *iodine* tincture. In obstinate cases it may be necessary to resort to a 25 per cent aqueous solution of *ichthyol*. Care must be observed in using *ichthyol*, since it is likely to provoke an acute dermatitis.

ANIMAL PARASITES.

The animal parasites with which children are most likely to be infested are *pediculi*, or *lice*, and the *itch mite*, or *scabies*.

Pediculosis: Lice.—Pediculosis is a contagious animal parasitic affection in which the body is infested with lice. These set up both primary and secondary lesions.

Symptoms.—In infants and children pediculosis is, as a rule, confined to the scalp. The uncleanly are mostly attacked. These parasites attack the scalp, causing much itching and scratching; escape of serum and purulent fluid occurs, forming crusts. The hairs become matted together; scratch-marks, pustules, excoriations and furunculi contribute to this unsightly condition. The cervical glands become secondarily enlarged.

Occasionally an eczematous condition of the scalp accompanies *pediculus capitis*. Pediculi are found both upon the scalp and the hairs. The nits are usually upon the hairs. The term *plica polonica* has been applied to an aggravated state of lousiness, where living and dead lice and their nits have matted the hairs together, an offensive odor arising from the decomposing pus and crusts. Severe inroads are in some instances made upon the general health, traceable to the annoyance coincident to incessant itching and scratching.

Diagnosis.—The detection of pediculi and their nits, together with their resulting secondary changes, will at once establish the diagnosis.

Treatment.—Kerosene oil is the best remedy with which to kill the parasites and their ova. The kerosene, or petroleum

oil may be mixed with olive oil and some balsam of Peru added. This mixture is less irritating and more efficacious than the plain oil. A good formula is four ounces of petroleum, two ounces of olive oil and half an ounce of balsam of Peru. It should be applied freely, and the scalp subsequently covered with a muslin or oil-silk cap. On the following day the head should be shampooed with soap and water followed by the liberal application of diluted vinegar, which dissolves the nits. This procedure may have to be repeated a number of times before a cure is completed. Should eczema of the scalp be present it must receive suitable treatment.

Scabies; Itch.—Scabies is a contagious animal parasitic disease of the skin, in which *acarus scabiei* is the exciting cause of the lesions.

The male itch-mite rarely burrows beneath the epidermis. The female, however, penetrates deeply, making minute tunnels, which serve as a habitat. The acarus selects those regions where the skin is tender, as the axillary and interdigital spaces, producing papules, vesicles, pustules, bullæ, wheals, infiltrations, furuncles and crusts. Scabies is spread by direct contact and is chiefly encountered among the poorer classes who live in overcrowded households and under conditions making personal cleanliness a difficult matter.

Pathology.—Inflammation of the papillary layer of the skin results from the presence of the acarus. Itching, which is usually intense, is a very distressing symptom. It is particularly severe during the sleeping hours, since the female acarus is most active when the patient is protected by the warmth of the bed-coverings.

Diagnosis.—The diagnosis of scabies should not be attended with any difficulty. However, cases in which secondary lesions such as papules and pustules and eczematous patches have resulted from scratching and infection are often mistaken for some other more serious condition. The presence of characteristic lesions, situated in the interdigital and other favorite

regions, associated with marked and distressing itching, should lead one to a positive opinion.

Pediculosis causes itching only of the parts attacked. Itching as a symptom of scabies is frequently referred to parts unattacked. In *pediculosis* the scratch marks are mainly confined to the shoulders, chest and waist-line and the itching is usually worse during the daytime. In itch, on the other hand, the itching is worse at night, and the hands, the flexor surface of the wrists and arms, the axillary folds and the genitals are affected. This characteristic distribution as well as the progressive character of the affection and presence of the burrows of the mites also differentiates itch from eczema.

Treatment.—The best parasiticide against the itch-mite is the *balsam of Peru* either used alone or in combination with a sulphur ointment. A drachm each of balsam of Peru and sublimated sulphur to three ounces of vaseline makes an efficient formula. The affected parts should be scrubbed with warm water and tincture of green soap after which the ointment should be thoroughly rubbed in. The treatment should be carried out night and morning for three days, after which a complete change of clothing and bed-clothes is to be made and the child given a hot bath. The clothes should be baked before putting them into the wash.

Dermatitis, if excited, may be controlled by discontinuing the use of the ointment and instituting appropriate treatment.

CHAPTER XIII.

DISEASES OF THE BLOOD.

The total amount of blood in the body of a child is somewhat less in proportion to the body weight than in an adult. Likewise the specific gravity is lower, the average being 1050 as compared to 1055 in adults. It bears a close relationship to the amount of hemoglobin, which is also proportionately low during infancy and early childhood. In the new-born, however, the hemoglobin percentage is high, but thereafter it rapidly falls, ranging between 55 and 85 per cent. Under ordinary circumstances 60 per cent may be considered the minimum for a healthy infant.

The *red corpuscles*, or *erythrocytes*, are most numerous at birth. Even during the period of infancy they remain relatively more numerous than in childhood and in adult life. They gradually decrease from six to six and a half million per cubic millimeter at birth to four and a half to five and a half million in early childhood, and the normal standard of five million is attained later in childhood. Fluctuations in the number of erythrocytes is, however, more common than in adults; even daily variations can be observed.

The form of the red corpuscle is variable in the new-born, and nucleated corpuscles (*normoblasts*) may be seen. The corpuscles also readily lose their hemoglobin, forming the so-called shadows of Silbermann. Variations in form, and the occurrence of nucleated red corpuscles later in childhood, are, however, always pathological, but these changes are observed in any severe type of anemia and are not of as serious significance as in adults.

The *leucocytes* are relatively more numerous than in adults. In the new-born a leucocytosis is present. According to Hayem there may be as many as 18,000 leucocytes to the cubic milli-

meter, but they fluctuate widely under slight influences, such as diet. A leucocyte count of from 10,000 to 12,000 during infancy may be considered normal. In childhood the number varies between 8,000 and 10,000, being slightly higher than in the adult.

The various forms of leucocytes are: (a) *Lymphocytes*, or small mononuclear cells, which are believed to originate from the lymphoid tissue. They are about the size of a red blood corpuscle and contain a single large nucleus which almost completely fills the cell. A narrow rim of strongly basophile, homogeneous or coarsely reticular cytoplasm surrounds the nucleus. During infancy the lymphocytes are the predominating leucocyte representing from 50 to 60 per cent of the differential count. During childhood there is a gradual decrease of the lymphocytes with a corresponding increase of polynuclear leucocytes. The lymphocytes are most markedly increased in lymphatic leukemia. An actual increase is also noted in many cases of rickets, and a physiological increase occurs after feeding. The lymphocytes are also relatively increased in tuberculosis. An actual lymphocytosis is observed in the early stage of whooping-cough.

(b) *Large mononuclear cells*, derived from the bone marrow and spleen. They are much larger than the preceding form and are not so numerous, constituting about 6 per cent of the different forms. In infancy the percentage is higher, while in the fetus they are most numerous. The nucleus is vesicular, does not stain as deeply as that of the small leucocyte, and at times has an indented, horse-shoe appearance, believed to be a stage of *transition* to the polynuclear form. The protoplasm is faintly basophile and may show a fine reticulum. On account of their light staining they are often spoken of as "hyaline cells." These cells are increased, as a rule, in conjunction with the lymphocytes, but they are especially increased in the so-called pseudo-leukemia of von Jaksch and in malarial fever. In malarial infection there is not only an absolute

increase in the large mononuclear cells, but also a relative increase over the small lymphocytes. They are also increased in measles, and in syphilis, tuberculosis and in typhoid fever, when these diseases become well established. A differential count of these cells, therefore, plays an important role in the diagnosis of obscure febrile affections. In the cases of malarial infection without much fever and without quinine history the polynuclears are diminished and the large lymphocytes much increased (Krauss, *Jour. Amer. Med. Ass.*, Oct. 22, 1904). This also holds good in recent cases. As some difficulty may arise in distinguishing between a large and a small lymphocyte, Krauss gives the following rule: "Class all cells the size of a polynuclear cell as small unless the protoplasmic margin is relatively large, and contains scattered neutrophile granulations, which stamps the cell as a large one."

(c) *Polynuclear leucocytes*, or *neutrophiles*. They are large leucocytes with several nuclei connected by threads, therefore they are also called "polymorphonuclear." The nucleus takes the basophile stain while the protoplasm is neutrophile and contains distinct granulations. They are the most numerous of all leucocytes, excepting in infancy, constituting from 60 to 65 per cent in children and as high as 70 per cent in adults. In the new-born the polynuclear leucocytes represent about 63 per cent of the white corpuscles and they rise to 70 per cent in the first forty-eight hours. After that a rapid destruction of these corpuscles takes place and they fall to about 35 per cent. They are rapidly increased in infectious diseases, acting as phagocytes. These cells form the pus cells in all active suppurative processes.

(d) *Eosinophile leucocytes* are large, round, polynuclear cells containing coarse, granular bodies which stain deeply with eosin. Their affinity for this stain gives them their name. Normally but 2 to 4 per cent are encountered, but in leukemia there is both a relative and an absolute increase. An eosinophilia is observed in children infested with intestinal worms and is a fairly constant finding in asthma.

(e) *Myelocytes*, or *Markzellen*, being so named from their origin in the medullary cavity of long bones. They are never found in the blood under normal conditions. They are several times larger than a red blood corpuscle, and have a single nucleus that stains but faintly. The protoplasm contains neutrophile granulations. They are found in spleno-medullary leukemia in conjunction with an increase of eosinophiles, and in severe secondary anemias.

(f) *Mast-cells* are variously sized leucocytes, either mono- or polynuclear, their protoplasm containing strongly basophile granules. They are found in a small proportion in normal blood, but in leukemia, and especially in the secondary anemias of childhood, they are considerably increased.

The following laboratory methods are essential for a proper clinical study of the blood:

1. The determination of the *percentage of hemoglobin*. This may be performed roughly with the use of the Tallquist scale. A drop of blood is collected upon a piece of unglazed paper and its color compared with the scale of the Tallquist hemoglobinometer. The results are only approximate but the *bleeding time* can be estimated at the same time that the color of the blood is noted. A more accurate instrument for careful clinical work is the Dare hemoglobinometer. With this instrument undiluted blood is used, the blood being allowed to flow into a small chamber between two plates of glass. The color of this film of blood is then compared with a color scale made from a circular disc of colored glass. The scale is turned until the colors correspond and the percentage of hemoglobin can then be read off at the side of the instrument.

2. The determination of the *number of red corpuscles*. For the purpose of counting the red corpuscles the hemocytometer of Thoma-Ziess is used. This instrument is supplied with a pipette graduated to hold one cubic millimeter of blood and one hundred millimeters of diluting fluid. A one to two hundred dilution is usually employed excepting in severe types of anemia.

The blood is thoroughly mixt with a solution corresponding to the blood serum in density (Gower's solution) and a drop is then placed upon the counting chamber which is ruled off into squares so that the corpuscles can be readily counted and the number in a cubic millimeter accurately estimated. When a one to two hundred dilution of the blood has been made five large squares of the counting chamber, each containing sixteen smaller squares, are counted and four ciphers added to the total number of cells counted. The result represents the number of red corpuscles in one cubic millimeter of blood.

3. The determination of the *white corpuscles* is carried out on the same principle, but, as they are less numerous, a larger pipette, giving a dilution of one to ten, is employed. Usually a one to twenty dilution is used to facilitate counting the leucocytes. With a one to twenty dilution the number of leucocytes in a square millimeter are counted and this number is multiplied by two hundred. The result is the number of leucocytes in one cubic millimeter of blood. A 3 per cent solution of acetic acid is used as a diluting fluid. This destroys the erythrocytes and renders the leucocytes more conspicuous.

4. The *differential count* of the leucocytes is performed by preparing a thin film of blood, and staining the same with Wright's stain so that the various types of white corpuscles can be identified. Two hundred leucocytes are counted and classified and the percentage of the different types estimated. The film is best made by collecting a small drop of blood on a square cover-glass, covering the same with another cover-glass, and then carefully drawing the slips apart after the blood droplet has run out into a thin film. The spreads are dried and then stained with Wright's stain.

5. The microscopical appearance of a fresh drop of blood is of great importance for diagnostic purposes. The shape and size of the red corpuscles, the absence of rouleaux formation, the presence of nucleated red corpuscles, the presence of parasites (plasmodium of malaria), must all be taken into consideration.

6. The *Specific gravity* is obtained by floating a drop of blood in a mixture of chloroform and benzol of 1050 to 1060 specific gravity. A drop of blood is allowed to fall into a test tube containing ten cubic centimeters of the fluid, and, according as it drops to the bottom or floats on the surface, chloroform or benzol is added. When it remains suspended in the fluid the specific gravity of the latter is taken, it corresponding to the specific gravity of the blood drop. For a fuller description of the instruments, technique and methods of blood study the standard works on Clinical Diagnosis should be consulted.

ANEMIA.

Clinically two varieties of anemia are encountered, namely *primary* and *secondary*. The latter form is far more common and is usually less serious in its clinical aspect than a primary anemia. *Primary anemia* is a manifestation of a developmental defect in the blood making organs as in *aplastic anemia*, or of some unknown etiological factor which may either cause an increased destruction of the blood elements or interfere with the formation of normal, mature blood elements which are necessary to replace those destroyed in the spleen and tissues. *Secondary anemia* is a reduction in the amount of blood from hemorrhage or an impoverishment of the blood resulting from malnutrition and unhygienic surroundings. It frequently develops as a sequela of an acute infectious disease and is prominently associated with tuberculosis, rheumatism and syphilis. Chronic intestinal indigestion and intestinal parasites may be the cause of a secondary anemia of severe type. A diet deficient in iron and other mineral salts and perhaps vitamins may also lead to a secondary anemia.

There is a strong tendency for the blood to revert to the embryonic type whenever an increased effort to replace the blood elements becomes necessary and so it is common to find nucleated red corpuscles and myelocytes in the infantile anemias. Enlargement of the spleen is also frequently encountered as well as

a leucocytosis. In rickets, for example, various types of anemia may be encountered, ranging in severity from a slight reduction in the number of red corpuscles and the percentage of hemoglobin to a severe type with pronounced pallor, reduction of red cells, the presence of nucleated red corpuscles, a leucocytosis with myelocytes and enlargement of the spleen. Clinically the majority of cases of anemia in infancy and childhood are secondary and there is usually a nutritional disturbance, a history of improper feeding or a disease like rickets, scurvy, tuberculosis, etc. to account for the anemia. There is a rare form of anemia occasionally encountered in infants which is evidently a primary disturbance in the blood making organs; this is known as *pseudo-leukemia of von Jaksch*.

The following clinical classification for the secondary anemias is given by Morse:

Mild anemia, characterized by trifling reduction in the hemoglobin percentage and number of erythrocytes and absence of abnormal changes in the blood elements.

Severe anemia with pronounced diminution of hemoglobin and erythrocytes, together with changes in the size and shape of the corpuscles and the presence of normoblasts, or nucleated red blood corpuscles.

Anemias with leucocytosis are usually associated with more pronounced reduction in hemoglobin and red corpuscles than anemias without leucocytosis (Da Costa). Normoblasts and deformities in size and shape of the erythrocytes are encountered in these cases.

CHLOROSIS.

Chlorosis is a form of primary anemia which is seen most frequently in girls at the time of puberty, but it is not necessarily confined to this period of life nor to the female sex. Of the *etiology* nothing positive is known excepting that unhygienic surroundings, improper or insufficient food, lack of fresh air and sunshine, emotional disturbances and obstinate constipa-

tion are frequently intimately associated with the development of chlorosis. The heart and larger blood-vessels have been demonstrated by Virchow as under-developed in many instances.

The *symptoms* of chlorosis may make their appearance rapidly, or the disease may not be suspected for a long time until pallor and the characteristic greenish tint of the skin, on account of which it is popularly known as "green sickness," give a clue to the existing ill-health. The child complains of headache, and displays an aversion to mental or physical exertion of any kind. Exercise brings on dyspnea and palpitation, while the headache and languor are responsible for the indifference to both work and play.

The appetite is poor, and in many instances becomes perverted, so that the patient craves chalk, slate-pencils, coffee-beans, etc., which are apparently enjoyed. Indigestion and constipation are troublesome symptoms, and their correction materially hastens the cure.

In young girls, menstrual derangements are inseparably associated with chlorosis. Thus, scanty menstruation or amenorrhea are almost invariably encountered in these cases; likewise, dysmenorrhea and leucorrhea are common. Improvement in the chlorotic state results in prompt improvement of these conditions.

The red corpuscles are but slightly decreased in number, but there is a pronounced deficiency of hemoglobin, giving the individual corpuscles a noticeably pallid appearance. The disturbance is, therefore, primarily in the blood-making organs which are incapable of making corpuscles of a normal hemoglobin content.

Edema tends to develop about the ankle joints, and many patients present a puffy, fat appearance, indicating a hydremic state, with sluggish return circulation. The degree of anemia can be roughly estimated by the appearance of the palpebral conjunctiva, the lips and the matrix of the nails, but in order to follow the progress of the case accurately we should make

regular hemoglobin estimations with the Dare hemoglobinometer.

The *prognosis* of chlorosis is favorable, and it usually responds promptly to treatment, although there is a liability to relapses. The chlorotic child is probably more susceptible to tubercular infection than a normal individual and should therefore be carefully protected against exposure to this disease.

PROGRESSIVE PERNICIOUS ANEMIA.

This form of primary anemia is a rare disease, and is more seldom seen in children than in adults. The *etiology* is obscure. Birch-Hirschfeld advances the infectious theory, owing to the presence of tissue destruction and retardation of blood coagulation; others hold to the theory of increased hemolysis, and again others to decreased hemogenesis. Stengel (*Medical News*, Oct. 20, 1900) expresses the view that pernicious anemia is undoubtedly a disease resulting from the rapid destruction of red blood corpuscles, for the compensation of which the blood-making functions prove inadequate; and, further, that the source of the hemolytic agents is the gastrointestinal tract.

The anemia resulting from intestinal parasites is very difficult to distinguish from pernicious anemia, showing the great liability for error and the difficulty with which a study of the disease is beset, as so many factors are capable of inducing pronounced anemia. In eighteen cases seen by Osler (*Amer. Text-Book of Practice*) there was no appreciable cause for the disease. Henoch (*Vorlesungen ü Kinderkrankh.*) saw two children in the same family die of this disease, no cause being ascertainable. Ewing thinks that any case of pronounced, progressively-increasing anemia in which the blood contains megaloblasts and a considerable proportion of megalocytes with increased hemoglobin, while the lymphoid marrow shows marked hyperplasia of peculiar type, should be considered one of pernicious anemia, regardless of the immediate exciting cause.

Even in the gravest secondary anemias these changes are rare, but in early life the changes in the blood are so pronounced in anemia that they are difficult to interpret. The frequency of pernicious anemia in childhood, therefore, is still a question.

The *symptoms* are those of a gradually increasing anemia. Loss of flesh may be absent. Edema and hemorrhage may supervene. The skin assumes a characteristic lemon-yellow tint. Anorexia, vomiting and other digestive disorders accompany the condition. The patient eventually dies from exhaustion, although remissions, leading one to believe that the case is recovering, frequently occur. The blood changes are the same as found in the adult as far as pronounced oligocythemia and nucleation and deformities of the erythrocytes are concerned, but the blood often fails to show the high color index and the prevalence of megaloblasts and of megalocytes that are accepted as diagnostic of the disease in adults (Da Costa).

Treatment.—The hygienic management of cases of anemia is of first importance. In chlorosis it is important to overcome constipation; this is best accomplished by means of diet and enemata. Fruits and fresh vegetables, many of which are rich in iron (notably spinach), are very beneficial. For anemia in general it may be said that the most nutritious and most digestible form of food is to be selected. The digestion is usually impaired and an achylia gastrica may be present, for which reason it is often desirable to administer digestive ferments, such as pepsin or papain, or small doses of dilute hydrochloric acid.

Milk is an ideal food in all forms of anemia, and chlorotic subjects may drink of it freely, even between meals. Eggs are also very beneficial, being easily digested, and their yolk contains a large percentage of iron. There is some risk in using raw beef but meat is usually not well digested by these patients unless given rare. Beef-juice is better for young children than the meat itself.

Where exhaustion is a prominent symptom, rest instead of

exercise should be prescribed. Absolute rest in bed, with massage and liberal feeding, will accomplish more in such cases than exercise, which only adds to the exhaustion and tissue breakdown.

The following remedies are the ones most useful in the various forms of anemia:

Belladonna.—In *chlorosis*, when there is violent palpitation, throbbing headache, great weariness and desire to sleep in the afternoon, debility. The symptoms of *belladonna* are very similar to those of *ferrum*, especially the palpitation, dyspnea and rush of blood to the face, alternating with paleness; but there is not that intense anemia and persistent debility, gastralgia, vomiting, amenorrhea and anasarca indicating the latter remedy.

Ferrum is seldom of use elsewhere than in *chlorosis* to which it is strictly homeopathic, as indicated by its symptomatology. Here it has gained universal reputation, and even its empirical use in large doses is frequently of great benefit. No doubt such remedies as *pulsatilla*, *nux vom.* and *spigelia* owe their usefulness in chlorosis to their influence upon the alimentary tract, and when they are indicated the use of *iron* is not always necessary for the cure, as sufficient iron should be absorbed from the food to supply the blood with all that it requires.

In pernicious anemia and in the secondary anemias iron is of little value as it does not act upon the hematopoietic organs. Many preparations of iron are in vogue, each form having its ardent advocates. *Ferrum reductum* in the first decimal trituration is one of the most reliable preparations; the *oxalate of iron* finds great favor with many of the British homeopaths in chlorosis. *Blaud's pill* is the best form in which to give iron in large doses.

Graphites.—*Chlorosis*, tendency to obesity, sluggish circulation and anemia, with general coldness; delayed or scanty menses, obstinate constipation; sad, tearful disposition.

Natrum mur.—*Chlorosis*, obstinate cases, fluttering of the heart, craving for salt.

Nux vom.—*Chlorosis*, gastric derangements, constipation, irritability, prostration; languid, especially morning on rising from bed; perverted appetite.

Pulsatilla.—*Chlorosis*, great weakness and sluggishness of the circulation, manifesting itself as chilliness; coldness and paleness of face, relief in open air. Anorexia, nausea, palpitation of the heart and dyspnea, sharp pains about heart (compare also *spigelia* and *cactus*, both of which are indicated by their cardiac symptoms), amenorrhea, leucorrhea; sad tearful disposition.

Other important remedies in chlorosis are *calc. c.*, *helonias*, *sepia* and *sulphur*.

China is a most valuable remedy in anemia developing after hemorrhages, chronic diarrhea, long-continued suppuration, and in all mild forms of idiopathic anemia as a "tonic," given in doses of two to three drops of the tincture, three to four times daily.

Arsenicum corresponds more closely to the *pernicious forms of anemia* than any other remedy, and is also indicated in the anemia of malaria and of Bright's disease. Its indications are excessive debility, edema of the ankles and eyelids, cardiac weakness and dyspnea, gastric irritability.

Phosphoric acid and *silicea* are useful in the anemia of debilitating diseases, such as typhoid fever, following well after *china*.

Mercurius is specific in the anemia of syphilis.

Kali carb. corresponds to a vitiated state of the *blood plasma*. Farrington refers to its ability to produce anemia, and recommends it for the blood poverty after severe or protracted diseases. The following symptoms are recorded in Hering's *Condensed Materia Medica*: "Vertigo, congestion to head with throbbing and humming. Swelling like a bag between upper eyelids and eyebrows. Palpitation in spells, taking his breath; stitches about heart; weak, irregular pulse. Arms go to sleep. Swelling of feet to ankles. Anemia, with

great debility; skin milk-white; muscles weakened, especially the heart." Our claims for the value of this remedy in anemia have been substantiated lately by old school therapeutics. Denstedt and Rumpf (*Therapeutische Monatshefte*, March, 1901) demonstrated that in pernicious anemia the blood gave a high percentage of water and sodium chlorid and a great reduction in the percentage of iron and potash. Accordingly, *potash salts* were administered in several such cases, both by mouth and infusion, with marked improvement. It seems that the death of the corpuscles depends upon the abstraction of its potash, and *potash*, therefore, has the same specific relationship to degenerative changes in the corpuscles that *iron* has to hemoglobin poverty in the corpuscles (chlorosis) and *arsenic* to the stimulation of the blood-making organs.

LEUKEMIA; PSEUDO-LEUKEMIA; SPLENIC ANEMIA;
HODGKIN'S DISEASE.

The varieties of anemia described under the above titles present as their most characteristic features permanent leucocytosis and splenic enlargement.

Their differentiation presents many points of difficulty, which can only be definitely settled by careful hematological examinations. They are seldom encountered during childhood, excepting the form known as *anemia infantum pseudoleukemia von Jaksch*. They all present an unfavorable prognosis.

Leukemia may affect persons of all ages, but it is rare during childhood. Mossa has collected a series of twenty-seven cases in children, but he admits that a large number of these were undoubtedly not cases of true leukemia. Da Costa collected ten cases, in all of which the diagnosis was confirmed by the examination of the blood.

The *symptoms* are anemia, pronounced pallor, distended abdomen, with enlargement of the spleen, and tenderness. The lymphatic glands may be principally involved, as in the *lymphatic variety*, or the spleen and marrow, in the *spleno-*

medullary variety. In the *lymphatic variety* the lymphocytes are markedly increased, sometimes the large, at other times the small mononuclear cells predominating. The polynuclear cells are relatively decreased. In a case coming under my notice the polynuclear cells had almost entirely disappeared from the blood, the blood-count giving five thousand leucocytes, mostly lymphocytes. The erythrocytes are diminished and a few normoblasts may be present. In the *spleno-medullary variety* there is a relatively small increase in the lymphocytes, but myelocytes are found in great numbers in conjunction with an increase in the eosinophile cells.

The disease assumes a progressively downward course, usually terminating in general edema, hemorrhages and exhaustion. At times it is febrile, simulating an infectious disease, and runs an acute course. This is more likely to occur with lymphatic than with spleno-medullary leukemia.

Hodgkin's disease presents enlargement of various groups of the lymphatics; enlargement of the spleen and liver; fever of an intermittent type, and progressive anemia and leucocytosis. The leucocytes are only moderately increased in numbers and abnormal elements (myelocytes and an increase in eosinophiles) are not present. The cervical and axillary glands, or those situated near by, are usually the ones first affected, other groups eventually becoming implicated. They do not, however, tend to break down, this being a strong point of differentiation between Hodgkin's disease and tuberculous adenitis. The course is chronic, and the prognosis is always unfavorable.

Anemia Infantum Pseudoleukemia von Jaksch is a disease of childhood, usually seen before the second year. The *etiology* is obscure. Several cases may occur in the same family. It was first described by v. Jaksch, its characteristics being: Occurrence in infancy; oligocythemia and oligochromemia; permanent leucocytosis; marked splenic enlargement, and at times lymphatic enlargement. The liver is but slightly enlarged, a clinical distinction between this disease and leukemia. The

prognosis is more favorable than in the latter disease, but many cases terminate fatally. The term *splenic anemia* has been applied to a class of cases similar in all respects to pseudoleukemia but without a leucocytosis.

The development of pseudoleukemia is one of progressive pallor, failure in general health, digestive disturbances, and at times slight pyrexia. The anemia is very noticeable, and palpation reveals an enlarged spleen. No doubt many cases described as pseudoleukemia are in reality a severe type of secondary anemia. Such an anemia is at times encountered in infants with rickets.

Treatment.—Homeopathic literature on these affections is meagre. Of our writers, Gilchrist enters most extensively into the subject in an article upon "Leucocythemia" (*Arnâ't's System of Medicine*), in which he also reports a case of Dr. Gaylord's represented as leukemia, which, however, should be classed as a case of anemia infantum pseudoleukemia. The patient was an infant of six months, anemic from birth, living in a malarial district. There was leucocytosis and splenic enlargement. *China* 2x and an occasional dose of *ferrum* resulted in a cure. Dr. Broadbent (*Hom. Review*, vol. xxi) recommends *phosphorus* as the most appropriate remedy in leukemia.

Gilchrist believes *china* and *phosphorus* to be the most closely related remedies to the disease. The old school depends upon *arsenic* in conjunction with *iron* and *cod liver oil* in leukemia, pseudoleukemia and Hodgkin's disease, although no claims for cures are made. The *X-ray* and *radium* have proven beneficial in Hodgkin's disease. Koplik has used *ichthyol* with some success in leukemia. Owing to its strong homeopathic relationship to rickets I should look upon *phosphorus* as the most appropriate remedy in the so-called "splenic" and "pseudoleukemic anemias."

HEMOPHILIA.

The subjects of hemophilia are commonly known as "bleeders," from the tendency to profuse and often uncontrollable hemorrhages which this form of constitution presents. The disease is hereditary, and the mode of transmission is a clear demonstration of atavism through the female, as hemophilia rarely occurs in females, being transmitted by the daughters of bleeders to their male offspring.

The *pathology* of hemophilia is not quite clear. In some instances it would seem to depend upon an abnormality in the walls of the small blood-vessels, and in others upon a delayed coagulation of the blood. The peculiarity which some cases present of only bleeding excessively in certain localities would favor the first mentioned explanation. According to Addis the cause of the delayed coagulation is a deficiency of prothrombin in the blood. A calcium deficiency has also been noted. The blood platelets are not diminished but there is a delay in their blood-clotting function.

The diathesis usually develops early in childhood, by the end of the first dentition period, when an accidental cut or injury first attracts attention to this tendency. Beside the danger of hemorrhage from a traumatism or an operation, there is as great a one from spontaneous hemorrhage such as epistaxis, hematemesis, hemoptysis, hemorrhage from the mouth, intestines, urethra, etc. Injuries without destruction of continuity of the skin are followed by profuse bloody effusions into the subcutaneous structures.

The hemorrhagic diathesis cannot be recognized until a hemorrhage has taken place, and the subjects are usually healthy-looking, characteristically supposed to have blonde or reddish hair, blue eyes, and a fair, transparent skin. There is a strong tendency to joint-affections of a painful type, which may resemble rheumatism of the larger joints closely. When a single large joint is involved in a child it is frequently mistaken

for a tuberculous lesion. A hemorrhage may be preceded by an attack of arthritis or circulatory disturbances, such as oppression, palpitation, and rush of blood to the head.

The *prognosis* is always grave, one half of the cases dying before the seventh year. As there is a tendency to outgrow the condition, the prognosis becomes more favorable with advancing years. There seems to be no unfavorable effect upon the functions of menstruation and parturition in female bleeders.

Treatment.—Powers (*Surgical Diseases of Children*) advises against the use of styptics in hemophilia, as they are always useless. The application of fresh blood to the wound has acted successfully (Bieudwald). The inhalation of carbonic acid gas (Wright, *British Med. Jour.*, 1894) has a decided influence over the epistaxis, which may also require plugging of the nares. Supra-renal extract is a most powerful styptic and less objectionable than tannin or perchloride of iron. Gelatin is highly recommended by some surgeons but is not reliable. The local use of cephalin (McLean) has proven most useful. Fresh human serum may be given intravenously at the same time.

As a constitutional remedy *phosphorous* corresponds most closely to the condition. The *lime salts*, given over a long period of time, may prove helpful in improving the bleeding tendency.

PURPURA.

The term purpura includes a variety of affections characterized by the development of reddish macules of varying size upon the skin due to spontaneous hemorrhage.

It may occur *symptomatically* after the administration of certain drugs (*iodides, quinine, potassium chlorate*, etc.); in the course of certain of the infectious diseases, notably in septicemia, cerebro-spinal meningitis, small-pox, septic endocarditis, and sometimes in measles; and as a result of cachexia, mechanical and nervous disturbances. *Primarily* it is observed

in the following clinical forms: *Purpura simplex*, *purpura rheumatica*, *Henoch's purpura*, and *purpura hemorrhagica*. Before classifying a case as one of purpura it is important to exclude any of the above mentioned conditions in which the skin hemorrhages are but a secondary manifestation and also eliminate such diseases as hemophilia and acute lymphatic leukemia.

Purpura simplex is characterized by the appearance of crops of purpuric spots, mainly upon the legs, which may be accompanied by slight fever, articular pains and diarrhea. The spots are bright red in color, do not disappear upon pressure, and gradually fade to a purplish and later to a greenish or dirty-yellow shade, the course pursued by all purpuric lesions. The duration is about one week. A rheumatic history is often present.

Purpura rheumatica, or *peliosis rheumatica* (Schönlein), as the name implies, bears a strong relationship to rheumatism. The purpuric rash develops in conjunction with multiple arthritis. The onset is usually that of an atypical rheumatic fever: lassitude, fever, sore throat, articular pains, and in the course of a few days the rash appears, which may be associated with urticaria. It is more common in adults than in children.

Henoch's purpura is, according to Henoch's own description, a complicated clinical picture, in which vomiting, intestinal hemorrhage and colic are associated with the symptoms of purpura rheumatica (*Vorlesungen ü. Kinderkrankh.*). The prognosis of this variety is usually favorable, Henoch reporting six cases, with recovery in all, and Osler eleven cases, with three deaths (*Amer. Jour. of Med. Sciences*, Dec., 1895). The diagnosis is often beset with difficulty, especially when there are no external hemorrhages. The symptoms may be entirely abdominal and simulate appendicitis so closely that cases have frequently been operated upon for appendicitis. Blood in the stools will usually be found on investigation and an acute hemorrhagic nephritis is a frequent accompaniment of the pur-

puric condition. In typical cases the paroxysms of abdominal pain and bloody stools show a tendency to recur and joint manifestations may develop during the remissions.

Purpura hemorrhagica (*morbis maculosus Werlhofii*).—*Purpura hemorrhagica* is characterized by the development of extensive subcutaneous hemorrhages, bleeding from the mucous membranes and a pronounced secondary anemia. The blood shows a marked reduction in the platelets, which accounts for the spontaneous hemorrhages.

The spots may extend over the entire body, their size varying from that of a pin-head to fairly large blotches. The macules are often interspersed with vesicles, produced by circumscribed hemorrhages into the rete Malpighii. The cutaneous hemorrhages are followed by bleeding from the mucous membranes and internal organs, particularly from the kidneys. Hematemesis is also noted in some cases. The duration is from ten days to two weeks in favorable cases. Death may result from exhaustion, or from a cerebral hemorrhage.

Purpura fulminans is a variety of purpura hemorrhagica occasionally seen in children. It is characterized by rapidly-developing cutaneous hemorrhages, which may of themselves be the cause of death. If the patient survives gangrene and sloughing may occur in the affected parts.

Treatment.—In cases of simple purpura and in the rheumatic forms, the best results will be obtained by prescribing for the underlying constitutional condition. Such remedies as *bryonia*, *arnica*, *hamamelis* and *rhus tox.* cover the symptoms found in these cases.

In the hemorrhagic form *china*, *crotalus*, *lachesis*, *kali hydrojodicum*, *phosphorus*, *rhus venenata*, *secale* and *sulphuric acid* should be studied and differentiated.

CHAPTER XIV.

DISEASES OF THE NERVOUS SYSTEM.

The nervous system in infancy presents certain anatomical and physiological characteristics which are responsible for some of the peculiarities noted in the neurological conditions encountered at this time of life. The psychology of the child must also be taken into consideration in order to properly interpret some of the symptoms and nervous disturbances of childhood.

The brain of the newborn represents one quarter of the body weight; nevertheless, it is in an immature condition both anatomically and physiologically. Owing to its large size and great vascularity, and because the cranium is only a membranous covering at this time of life, it is very liable to injury during parturition. The most important feature of the infantile brain is the imperfect development of the higher inhibitory centres and the absence of myelin sheaths for the neurons comprising the pyramidal tracts. This explains the tendency to convulsions (eclampsia) observed in infancy and the heightened reflexes normally present at this time of life.

In young infants there is a physiological hypertonia of the muscles and the tendon reflexes are therefore exaggerated. The extremities also offer a certain degree of resistance to passive motion and it may at times be difficult to determine whether or not a spastic condition is present. The *knee-jerk* is most satisfactorily obtained when the infant is preoccupied in nursing or when its attention is attracted by some object of interest. The examiner should place the left hand under the knee, raise the same sufficiently to flex the leg on the thigh and then strike the patella tendon with the middle finger of the right hand. In older children it is sometimes more satisfactory to have the child sitting up with the legs hanging over the edge of the bed or chair.

The knee-jerk is exaggerated in lesions affecting the upper neurons, i.e., cerebral lesions. In lesions of the lower neurons, i.e., spinal cord and spinal nerves, it is diminished or abolished (poliomyelitis, diphtheritic paralysis).

Kernig's sign is found in meningitis (about 85 per cent of cases), and at times in cerebellar hemorrhage and in lesions at the base of the brain. It is a phenomenon of hypertonia of the flexor muscles of the legs. This condition was originally described as an inability to extend the leg upon the thigh when in the sitting posture, owing to tonic spasm of the hamstring muscles. When the dorsal decubitus is assumed, the leg can be straightened out, but if the thigh is now flexed upon the abdomen it again becomes impossible to straighten out the leg and a spasmodic resistance is noted in the contracted muscles. *Babinski's sign* is an alteration in the type of response of the plantar reflex, there being hyperextension of the great toe instead of flexion. It indicates a disturbance in the pyramidal tracts. This most valuable diagnostic sign in older children and adults is of no clinical value during infancy since the normal infant gives a plantar reflex quite similar to Babinski's sign. The explanation for this is most likely the lack of development of the myelin sheaths for the fibres of the pyramidal tract. After the infant has learned to walk the normal plantar reflex usually develops.

An important clinical fact to bear in mind is that the majority of brain lesions during infancy and early childhood are either cortical or basilar. Hemorrhage into the internal capsule is rare, the usual type of hemorrhage being pial in character.

The function of the cranial nerves is determined in the manner employed for adults, as far as that can be carried out. Strabismus is normally present in infants under three months. After that age the infant acquires ocular control and begins to use its eyes in a coordinated manner.

Motor paralysis is detected by observing whether or not the child is able to move its extremities. Inability to walk may be

due either to paralysis or to rickets (rachitic pseudo-paralysis). In the latter condition the child can move the legs, as tickling the sole of the foot will prove, but it is unable to stand or to walk. *Spasticity*, or "lead-pipe rigidity," is found in cerebral palsies, usually in association with impaired mentality.

The *mental development* is difficult to gauge in infancy. The early signs of amentia, or idiocy, are inability to support the head (normal at four months); amaurosis (amaurotic family idiocy); crying without cause, backwardness in grasping and holding objects; inability to nurse properly. Normally, a child should begin to walk after it is a year old and shortly after this time it should commence to repeat words and use them intelligently. According to West, a backward child would be normal were it of a younger age, while an idiot is abnormal for any age.

Reaction of degeneration.—By the "reaction of degeneration" is meant the electrical phenomena which take place in a muscle supplied with a motor nerve whose spinal ganglion cell has been destroyed, or, in fact, whose lower neuron has been affected at any point in its course. The reaction is distinctive and differs so markedly from the reaction obtained by the galvanic current in a normal muscle that it is a reliable diagnostic sign.

Briefly stated, the muscle loses its irritability to the faradic current, while the contraction with the galvanic current becomes slow and tetanoid in character, the main change, however, being that it first responds to the anodal closure with a gradually increasing current instead of to the cathode, as occurs normally. The reaction of degeneration is found typically in poliomyelitis anterior. It also occurs in progressive muscular atrophy and in multiple neuritis.

PSYCHOSES.

Night terrors and *morbid fears* are mental disturbances in which hallucinations of various kinds are developed in the child's imagination through fright, or through the suggestions resulting from the recital of ghost-stories and fairy-tales, or

from vicious threats. The rational explanation for a large number of cases of *frightened awaking from sleep* is, in my belief, a choking spell resulting from enlarged tonsils and adenoids or some other form of suffocative attack (nightmare). As many children with adenoids present these symptoms the throat should always be examined in such cases. Another common cause of *disorders of sleep* is gastro-intestinal irritation; but in these cases the symptoms are reflex in character, and do not approach the nature of a psychosis, as do the above. In neurasthenia and lithemia similar disturbances are observed. The idiopathic fears and terrors point to a highly neurotic form of constitution, and they may indeed be the forerunners of a more serious mental trouble. It should also be remembered that in epilepsy there may be a psychic equivalent for the convulsion and that a child with unusual mental symptoms may be an epileptic.

IDIOCY AND MENTAL DEFICIENCY.

Idiocy is mental deficiency in the extreme form. The idiot is a helpless, hopelessly stupid child who cannot care for himself and who cannot be taught to do anything intelligently. He may attain the mentality of an infant one or two years old but as a rule he does not show as good intelligence as a normal infant and he may be exceedingly abnormal in disposition and in his actions.

Mental deficiency is encountered in different degrees and a child may appear to be normal until a psychological examination has been made and shown it to be mentally backward. An individual whose mental development has been arrested at a stage corresponding to the mentality of a normal child of ten or twelve years is spoken of as a *moron*. Two types of idiocy are recognized, namely *primary amentia*, which is a mental defect due to an intrinsic cause (morbid heredity), and *secondary amentia*, due to extrinsic causes such as traumatism, infection or unfavorable environment.

In the *etiology* of idiocy hereditary transmission plays the most important role; of all mental derangements it is the one most frequently transmitted directly. A neuropathic family history; parental epilepsy or insanity; consanguine marriages; alcoholism in the parents; worry or fright of the mother during pregnancy, are etiological factors of less importance. The pathological lesions responsible for idiocy are either present at the time of birth, having developed *in utero*, as in primary amentia, or they may develop after birth as in the case of traumatic, inflammatory, epileptic and paralytic idiocy.

The following classes of idiocy are recognized by Ireland:

Genetous idiocy, cases which cannot be traced to any known specific disease, and whose pathology cannot be properly diagnosed until after death. The condition of mental deficiency is present before birth. There is usually a history of imbecility in one of the parents or in the family of one of the parents. Goddard has shown that feeble-mindedness is hereditary and is transmitted according to the Mendelian law, like any other character. It is a Mendelian recessive character.

The expression of the idiot is generally good-natured but stupid; the head is not necessarily small, although irregular in shape. Stigmata of degeneration are usually present. The early symptoms of genetous idiocy are constant sleeping in early infancy and absence of interest and attention to its surroundings, inability of the infant to suckle well, a feeble grasp, failure to react to sensory impressions and sight, and backwardness in walking and talking. The occurrence of such symptoms in the presence of a neuropathic heredity should always arouse our suspicions.

Amaurotic family idiocy is a clinical type described by Sachs. It occurs in Jewish families, the child being apparently normal at birth but gradually sinking into a state of complete idiocy with loss of sight. The outcome is fatal.

Microcephalic and *hydrocephalic idiocy* are forms of idiocy which are usually congenital, like genetous idiocy, although hydrocephalus may not develop until after birth.

Epileptic and *paralytic idiocy* belong to the acquired forms of the disease, developing in consequence of some other disease of the nervous system.

Paralytic idiocy depends upon destruction of cerebral substance from lesions which may have developed either before birth (congenital idiocy) or after birth (acquired idiocy). In these cases there is frequently sufficient asymmetry of the brain present to produce noticeable inequality of the skull. Hemiplegia, more or less complete, the arm usually more affected than the leg, or diplegia are the accompanying conditions. Frequently the child's mentality is less impaired than mere appearances would indicate. The physical handicap makes the mental deficiency more apparent than real and much can be accomplished in these cases by proper training.

Inflammatory idiocy includes those cases following meningitis or some of the infectious fevers (post-febrile insanity); also idiocy depending upon atrophy and hypertrophy of the brain, the result of inflammatory changes.

Sclerotic idiocy presents sclerosis with atrophy of the brain, diffuse sclerotic changes, and glioma with sclerosis (Wilmarth, *Alienist and Neurologist*, Oct., 1890). As predisposing causes are mentioned the tuberculous diathesis, neuropathic heredity; alcoholism. Accidents to the mother during pregnancy and traumatism to the child's head during or after birth are exciting causes.

Syphilis no doubt is responsible for many cases of idiocy. It is perhaps more common to find mental and physical inferiority and certain neurological conditions resulting from inherited syphilis than imbecility. "Our verdict at the present time is that, as regards the causation of mental defect, syphilis is a sufficient factor in itself, and often has a deciding influence when there is a morbid heredity or other unfavorable factors" (Shuttleworth and Potts, *Mentally Deficient Children*). The pathological lesions which congenital syphilis may give rise to are "a meningitis, a hydrocephalus, an endarteritis, gummata,

juvenile tabes, and juvenile general paralysis of the insane" (Batten).

Traumatic idiocy results from pathological changes in the brain, induced by a destructive injury. A certain amount of inflammatory action must always be taken into consideration in these cases, but the effects of the injury predominate over those of the inflammation. The traumatism is usually a birth injury (see *paralytic idiocy*).

Deaf-mutism is a form of mental deficiency through deprivation of the sense of hearing. If deafness is acquired after the seventh year the child usually escapes mutism.

Treatment.—Medical treatment is of no value in improving the child's mentality excepting perhaps in cases of syphilitic origin. The chief factors in the care of the feeble-minded child are careful nursing, or "mothering," improving the child's physical condition by the use of a constitutional remedy, proper diet and the correction of such defects as nasal obstruction (adenoids) and errors of vision and finally skilful training. During the first six years of life the backward child is still practically in its infancy and at this time its physical welfare alone need be looked after. Some training, such as the acquisition of habits of bodily cleanliness and learning to feed itself may be begun. After the sixth year, however, it should be placed in the hands of a tutor who has had special training in the education of feeble-minded children. The best results are usually obtained when the child is sent to a special school because it here receives the advantages of class work and also the benefit of coming under the care of highly trained specialists in this work.

MONGOLIAN IDIOCY.

Mongolian idiocy is a form of primary amentia not of hereditary origin but occurring as a result of failing procreative powers on the part of the parents. Mongols often represent the last born child of a large family in which case the mother is nearing middle life or has become exhausted from too frequent

childbearing. Extreme age of the father may also be noted in the history of the case. Shuttleworth and Potts refer to the mongol as an unfinished child, representing a phase of fetal life. There is no evidence of syphilis as an etiological factor. The sexes are about equally affected.

The mongol presents a characteristic appearance, his resemblance to the mongolian race giving him the name. He is undersized and physically frail; many mongolians die during infancy and few grow up to adult life. The head is round, or brachycephalic; the eyes are obliquely set and almond-shaped and an epicanthic fold is present. The nose is flat and the tongue is transversally fissured and has hypertrophied papillæ. The fissures, however, do not develop until the child is three to four years old. The tongue is not enlarged as in cretinism. The hands are broad and the fingers are short. The little finger often shows an inward curve of the distal phalanx. A congenital heart lesion is frequently encountered in these children. They are usually mouth-breathers but the removal of adenoids does not bring much relief from this condition since it is mainly due to the poorly developed nasal chambers and high arched palate and to lack of attention on the part of the child.

The mental and physical development is slow and never reaches beyond the mentality of a very young child. They do not walk until after the second year and are late in learning to talk. Their speech is always very elementary and usually difficult to understand. They may show great love for music, however, and they often show unusual powers of mimicry but they seldom can be taught to do more than the simplest things in life. Milder types, simply suggestive of the mongol, may prove more satisfactory from the mental standpoint.

Mongolism is frequently mistaken for *cretinism* and it is most important, both from the standpoint of prognosis and treatment, that the two conditions be not confused. In mongolism the child's abnormality exists from birth while cretinism does not show its characteristics until after the sixth month.

The slanting, close-set eyes; epicanthus; rounded, brachycephalic head give the mongol a very characteristic appearance quite different from the short, stunted, fat, pot-bellied cretin with his dull, apathetic face, large head, eyes far apart, thick lips and protruding tongue.

Treatment.—Mongolism differs from cretinism in that it does not respond to treatment with thyroid gland extract. Small doses of thyroid extract no doubt benefit some cases purely through the tonic effect of this substance on metabolism but the large doses which are frequently used do more harm than good. Careful nursing and training as recommended in the management of feeble-minded children applies to the mongol in every particular.

SPORADIC CRETINISM.

The clinical type of congenital hypothyroidism in which the pediatricist is particularly interested is known as *sporadic cretinism* because it occurs sporadically in our midst and bears a close clinical resemblance to the endemic form of cretinism which has been observed for years in certain mountainous regions of Europe. The pathology of cretinism is atrophy or absence of the thyroid gland. The results are dwarfing of both the physique and mentality of the individual together with signs of myxedema.

As a rule the infant is normal at birth since it receives sufficient thyroid secretion for its normal development from the maternal blood. After the sixth month, however, it begins to give indications of mental dulness and retarded development. The earliest symptoms are apathy, obstinate constipation with a large abdomen; the skin becomes harsh and dry. The fully developed cretin is very characteristic. He presents a dwarfed body, short legs and arms, sluggish mentality or idiocy, a large square head with eyes set wide apart, pug nose and pouting lips. The tongue becomes enlarged from myxedematous infiltration and the skin is loose and redundant from the same cause. The

abdomen is large and pendulous and there is usually an umbilical hernia.

The remarkable fact in connection with cretinism is the striking result which follows the administration of thyroid gland in these cases. There is a prompt improvement in the child's mental and general condition. It becomes bright, constipation disappears and it begins to grow and lose its unsightly features. Treatment, however, must be continued indefinitely or the child will sink back into its abnormal state. The earlier treatment is begun the better the outlook for mental advancement. The best results to be expected are that the child will be approximately normal; they usually remain backward to some extent although capable of acquiring a certain amount of knowledge. Two to three grains daily of the thyroid gland extract is usually necessary to keep the child in its best condition.

MENINGITIS.

Meningitis, or inflammation of the meninges of the brain, is usually encountered in the acute form during childhood and may be either *primary* or *secondary*. The chief forms of primary meningitis are acute (epidemic) cerebrospinal meningitis and tuberculous meningitis. The epidemic form of cerebrospinal meningitis is due to infection with the meningococcus intracellularis; it occurs sporadically as well as in epidemics but infection is spread by "carriers" and it is usually encountered during the winter months.

Secondary meningitis is a complication of one of the acute infectious diseases and it may, therefore, develop during the course of pneumonia, enteritis, influenza, typhoid fever and purulent otitis media. It usually runs the course of a *purulent meningitis*.

Serous meningitis, or **meningismus**, is an acute inflammation of the pia mater with resulting serous exudate. It is usually a secondary condition, developing in conjunction with some

acute disease, notably a gastrointestinal affection as a secondary infection of the meninges with germs of low virulence or it is purely toxic in origin. The symptoms are similar to those observed in other forms of meningitis although less pronounced. There may be convulsions and strabismus; infants present bulging fontanels while older children complain of headache. Stupor gradually develops and such symptoms as rigidity of the neck and Kernig's sign may be present.

Diagnosis.—The diagnosis of meningismus is based upon the presence of symptoms of brain irritation and pressure which accompany some acute infectious process, such as, infectious diarrhea, pneumonia or otitis media and which tend to clear up with recovery from the primary disease. Lumbar puncture is of the greatest help in the diagnosis. An increase in the amount of cerebrospinal fluid is found but the fluid is clear and there is no increase in its cellular elements or in its globulin content. Cultures made from the fluid are negative.

Treatment.—The treatment is chiefly that of the underlying condition. Lumbar puncture gives prompt relief, as a rule, from the cerebral symptoms. It is not uncommon to see the child come out of its stupor and to note a cessation of convulsions after the intracranial pressure has been relieved by a successful puncture.

When the cerebral symptoms dominate the clinical picture such remedies as *apis*, *belladonna*, *cicuta*, *helleborus*, *cuprum arsenicosum* and *hyoscyamus* suggest themselves and may be prescribed on their characteristic symptoms as given elsewhere. Whenever there is any doubt as to the diagnosis, particularly early in the case and during an epidemic of cerebrospinal meningitis the safest mode of procedure is to give an intraspinal injection of meningococcus serum. No harm can arise from this procedure and should the case later prove to be one of meningitis, valuable time in treating the case has been gained.

EPIDEMIC CEREBROSPINAL MENINGITIS; SPOTTED FEVER.

Epidemic cerebrospinal meningitis is caused by the *meningococcus intracellularis* which was discovered by Weichselbaum in 1888. It is a Gram negative diplococcus, found within the polynuclear cells of the spinal fluid. Infection takes place by way of the upper respiratory tract where the meningococcus is harbored and thence carried to the meninges by way of the blood current. The organism can be recovered from the nose and throat of infected individuals and from "carriers" of the disease.

Epidemics occur most frequently in the winter months. It does not spread rapidly and an epidemic may therefore extend over many months. Sporadic cases can probably be explained on the ground of contact with a carrier who has brought the infection from some distance. Young children appear to present a special susceptibility to the disease. Poverty, overcrowding and unhygienic surroundings are predisposing causes.

Pathology.—Rapidly fatal cases may show only the signs of severe cerebral congestion. A fully developed case, however, presents a typical purulent meningitis. Cases which have run a protracted course show thickening of the meninges; degenerative changes in the cerebral cortex; marked distention of the ventricles.

The pathological process is an exudative inflammation of the pia mater affecting chiefly the base of the brain and the posterior surface of the cord. The exudation into the cord is most pronounced in the dorsal and lumbar region. Effusion into the ventricles and into the pia mater of the cortex co-exists to a lesser degree. The cranial nerves are more or less involved according to the amount of exudate and pressure which is present. The auditory nerve is especially liable to involvement and permanent nerve deafness is a common after effect of the disease. The exudate is at first sero-fibrinous, soon becoming purulent. It is rich in polynuclear leucocytes. Some degree of cerebritis may co-exist.

Associated lesions that may be encountered are cutaneous hemorrhages (petechiæ); nephritis; bronchopneumonia; parenchymatous degeneration of the heart, liver and kidneys; arthritis.

Symptoms.—The disease is very irregular in its clinical manifestations and may either prove fatal within a few hours or run a long and protracted course. There are intermediate cases of moderate severity in which perfect recovery takes place. Epidemic cerebrospinal meningitis is the least fatal form of meningitis, but unfortunately many cases that recover are left with some permanent disability such as deafness, blindness, idiocy, paralysis.

A number of types of cerebrospinal meningitis are to be recognized, the classification being based chiefly upon the duration and severity of the symptoms. The invasion, however, shows a general resemblance in all cases. The onset is sudden, with either vomiting or convulsions, intense headache and fever, and soon the most characteristic symptom of the disease, namely, rigidity of the neck muscles and retraction of the head, makes its appearance.

In the *fulminating* form the onset is so sudden and overwhelming that the patient may succumb within a few hours.

These foudroyant cases usually prove fatal in the first few days. Deep coma develops early and is associated with retraction of the head and even opisthotonos. Strumpell refers to a class of cases with sudden and severe onset, similar to the fulminating form, which, however, abort in the course of several days and go on to complete recovery (*abortive form*). There are also *mild cases*, in which the entire clinical course is marked by slight development of the symptoms. A child thus affected may show the meningococcus in its cerebrospinal fluid and yet not appear to be seriously ill.

Protracted cases are not uncommon. The symptoms may extend over a period of from two to three months and the child ultimately recover. In these protracted cases the fever dis-

appears for several days and then recurs. Such cases have been designated "intermittent cerebrospinal fever" (von Ziemssen). A pronounced degree of emaciation develops in the case of this type. There is a protracted form of cerebrospinal meningitis which has been described in the literature as "*posterior basic meningitis*" thus giving the impression that it is a distinct disease. Posterior basic meningitis, or *chronic basilar meningitis* is, however, identical with cerebrospinal meningitis both in etiology and pathology. It is usually encountered in infants because the anatomical peculiarities of the brain at this time of life predispose to the exaggerated basilar manifestations.

Typical cases present the following symptoms:

The *onset* is sudden although such prodromal symptoms as headache, malaise, conjunctivitis and slight fever may be noted.

The initial symptoms point to the brain as the seat of the affection. There are vomiting or convulsions, sometimes a chill; intense occipital headache and high fever. To these symptoms stiffness of the neck is soon added and with the outpouring of exudate more or less disturbance of consciousness.

In infants bulging of the fontanel is to be noted. *Delirium* is a common symptom with older children. Complete stupor develops, although often the patient may be aroused, or lucid moments will alternate with the stupor. Fever and delirium may be present only at night, the child being rational during the day and able to sit up in bed and play with its toys (Koplik).

The headache may be so intense that we will observe the child knitting its brows and moaning with pain, while at the same time it is so deeply in stupor that we can neither arouse it nor get a response. Beside the cerebral manifestations we will observe marked hyperesthesia of the cutaneous surface, due to the irritation of the posterior nerve roots (spinal) by the inflammatory exudate. This hyperesthesia is most marked in the lower extremities.

Retraction of the head occurs earlier (within a day or two or even within a few hours) and is more pronounced and more

persisting in cerebrospinal fever than in any other form of meningitis (Heubner). As a rule, there is tenderness along the entire spine, which may be rigid or arched.

Disturbances in the functions of the cranial nerves are manifested as hyperesthesia of the sensory and irritation of the motor nerves. Thus photophobia, tinnitus aurium and disturbances of smell are encountered. Optic neuritis may develop with consequent blindness, and permanent deafness is one of the unfortunate sequelæ of the disease.

Spastic strabismus; irregular but reacting pupils; ptosis; spasm of the facial muscles and dysphagia are all to be observed. In contradistinction to tuberculous meningitis, there is more tendency to irritation and less to actual paralysis in cerebrospinal meningitis than in the former.

The extremities are rigid, the arms usually being flexed while the legs are straightened and resist passive movements. If, however, we flex the thigh upon the abdomen, or if the patient attempts to get up, spasmodic flexion of the leg upon the thigh takes place. This phenomenon is known as Kernig's sign. It is readily demonstrated by flexing the thigh upon the abdomen with the patient in the dorsal position, and then attempting to extend the leg on a line with the thigh. Kernig's sign is present in a large proportion of all cases of meningitis, but especially when the meninges of the cord are at the same time involved. It indicates irritation of the pyramidal tracts; Fraenkel (New York) thinks the phenomenon depends upon traction on the cauda equinæ, and he calls attention to the fact that we often can see the Babinski sign take place in the foot simultaneously with the occurrence of the Kernig.

The *cutaneous* manifestations are important, and we should not forget that the disease acquired its old name from the petechial rash that is present in about a third of the cases. In Osler's cases the rash was common. Fully one-half, if not more, present herpes labialis or facialis (Strumpell).

The *fever* is irregular and does not conform to any type of

temperature curve. The temperature is usually high in the beginning and there may be hyperpyrexia in the more severe cases. It is usually irregular and marked by remissions and even intermissions of several days at a time.

The *pulse* is rapid and the characteristic slowing observed in tuberculous meningitis is not encountered. In fulminating cases, however, it may be slow and irregular. This is a grave symptom. The *respirations* show nothing pathognomonic; exceptionally Cheyne-Stokes respiration occurs.

The *blood* shows a distinct leucocytosis.

The duration of the disease is usually from two to three weeks although complications may prolong the course while some cases tend to become protracted. The early use of Flexner's anti-meningococcus serum tends to materially shorten the course of the disease as well as to decidedly reduce the mortality.

Complications are observed in some cases, they are more common in some epidemics than in others. Aside from the complications on the part of the nervous system already referred to there may be clonic contractions and paralysis of individual extremities and occasionally unilateral paralysis. They appear to be due to various pressure conditions by meningeal exudates or possibly they owe their origin to variations in circulatory conditions, but at the autopsy a clear insight as to what has caused these paralyzes is by no means always obtained (Eichhorst).

Pneumonia and *arthritis* may occur as metastatic inflammations. The arthritis of cerebrospinal meningitis closely resembles acute articular rheumatism and may be associated with endocarditis.

As *sequelæ*, long-continuing nervous disturbances, such as vertigo; headache; loss of memory; neurasthenia are common. Permanent deafness, blindness, epilepsy, idiocy and chronic hydrocephalus may also be observed.

Prognosis.—Excepting in the mild and abortive cases the prognosis is grave, unless prompt serum treatment is instituted.

Before the time of Flexner's serum the mortality was about 60 per cent. Now it is about 25 per cent in cases treated with serum.

Diagnosis.—In fulminating cases that die before the clinical picture of the disease is developed, it is naturally impossible to make a diagnosis. When, during an epidemic, however, a child develops fever, vomiting or convulsions and rapidly goes into a state of coma, it is fair to surmise that we are confronted with a case of cerebrospinal meningitis. Should retraction of the head develop, the diagnosis is almost certain. We must, however, not forget that *pneumonia* may begin precipitately with marked cerebral symptoms. Careful exploration of the chest will decide the question under these circumstances. When pneumonia complicates cerebrospinal meningitis the pulmonary symptoms do not develop until later in the disease.

Osler has called attention to cases of *typhoid fever* beginning abruptly with delirium, headache, retraction of the head and high fever. If such a case dies early, differentiation is impossible unless fluid containing the meningococcus can be obtained from the spinal canal.

Differential diagnosis is mainly between cerebrospinal and *tuberculous meningitis*. In cerebrospinal meningitis the onset is more sudden; the fever is higher; retraction of the head occurs earlier and is more marked and the nervous manifestations are more irritative and less inclined to become paralytic in nature. The hyperesthesia of the skin and the petechial rash when present are strong, confirmatory symptoms. Then again, the presence of an epidemic is to be taken into consideration and in the case of tuberculous meningitis a tuberculous history may be obtained.

The examination of the *spinal fluid* obtained by means of a lumbar puncture, offers the most positive data for confirming the diagnosis. The fluid is increased in both diseases but not so markedly in cerebrospinal meningitis as in tuberculous meningitis. It is turbid and of a milky appearance while in tuber-

culous meningitis it is perfectly clear. Globulin is increased in both diseases and the reduction of Fehling's copper solution is diminished. In poliomyelitis the reduction of the copper solution is positive. The sediment consists of polynuclear leucocytes in great abundance and the Gram-negative intracellular diplococcus of Weichselbaum can usually be demonstrated.

Treatment.—The best results in the treatment of cerebrospinal meningitis are obtained by the use of Flexner's anti-meningitis serum. This is a polyvalent serum prepared by immunizing horses with the toxins and cultures of a variety of strains of meningococci. A recent refinement in the serum treatment of cerebrospinal meningitis is to culture the organisms found in the patient's cerebrospinal fluid and test out a number of sera against the culture in order to determine the one most suitable for the case. While this is an excellent mode of procedure to adopt in hospital work, still it offers difficulties for private practice where we must depend on a Board of Health serum or one of the commercial sera of good repute. As in the case of diphtheria, the earlier the serum is employed the better will be the results; therefore it is always wise to be prepared to give an intraspinal injection of serum at once, if the appearance of the cerebrospinal fluid corroborates the clinical diagnosis. If the further laboratory examination of the fluid verifies the diagnosis a second injection of serum may be given the next day and the injections repeated until the clinical symptoms have been controlled. The dose for an infant is 10 to 15 c. c. and for a child 20 to 30 c. c.

As an adjuvant in the treatment hot baths are of a decided value. They are indicated in the beginning of the disease and undoubtedly exert a sedative effect upon the nervous manifestations. Beginning with a temperature of 98° F. the heat can be increased daily by a degree, up to 105° F. One bath daily is sufficient, and as little handling of the patient as possible is to be advised on account of the suffering caused thereby. An ice-

bag to the head sometimes gives relief from the intense headache.

Of the greatest importance is the feeding of these cases. Extreme emaciation results unless we take advantage of every opportunity of getting sufficient nourishment into the child. Tube feeding may prove necessary in extreme cases.

Remedies.—In the early stages *belladonna* is the most valuable remedy, corresponding to the meningeal congestion both symptomatically and pathologically. *Bryonia*, *apis mellifica* and *cicuta virosa* are indicated when the signs of meningitis are more fully established. When toxic symptoms predominate over inflammatory, such remedies as *hyoscyamus*, *opium* and *helleborus* are more suitable. Cases with marked petechial eruption and of protracted character call for *arsenicum*, the snake venoms, and especially *rhus toxicodendron*. Cases with convulsions call for *cicuta virosa*.

Actea racemosa is useful for the pains and spasm persisting after the acute symptoms have subsided (Searle). There is intense occipital headache, like a bolt being driven from the nape of the neck to the vertex, felt with every pulse-beat; stiffness of neck; delirium.

Apis mellifica.—Sopor, interrupted by piercing shrieks; squinting; pupils dilated; retraction of head (stage of effusion).

Arsenicum.—Protracted and adynamic cases, intermittent type.

Belladonna.—High fever; convulsions; flushed face; photophobia; difficulty in swallowing; intense throbbing headache; delirium; vomiting; marked drowsiness.

Bryonia.—Bursting headache; apathy; child cries when it is touched or moved; arthritis or pneumonia.

Cicuta.—The toxicologic reports of this remedy show its pronounced action upon the meninges of the brain and cord, in which it sets up intense congestion with resulting convulsions. Various forms of paralysis may follow upon the convulsions. More or less disturbance of consciousness is associated. Dr.

Baker (*Trans. New York Hom. Soc.*, 1872) reported most promising results from *cicuta* after using it in an epidemic at Batavia, New York.

Cuprum aceticum has long been recognized as a potent remedy in meningitis, and was used with success by Dr. George Schmidt, of Vienna, for the cerebral symptoms accompanying the infectious diseases. Goodno considers *cuprum aceticum* the most generally useful remedy in cerebrospinal meningitis, giving it the preference over *cicuta* when cerebral symptoms predominate over the convulsive symptoms.

Gelsemium.—Early stages, chilliness, aching and prostration, photophobia, ptosis and squinting; occipital headache, with muscular soreness in the neck; remitting fever.

Helleborus.—Stupefaction, child bores its head into the pillow; suppression of urine, convulsions (serous effusion).

Hyoscyamus.—Muttering or wild delirium, unconsciousness, convulsions, pupils dilated, purplish rash. The temperature is not as high as in *belladonna*.

Kali hydrojod.—*Iodide of potash* is the remedy chiefly relied upon by the old school. There are some encouraging reports from its use, and it may be tried with advantage in cases not presenting marked symptoms for another remedy.

Opium.—Deep coma, pupils fixed, stertorous breathing, pulse irregular, inclined to be slow, clammy skin.

Rhus toxicodendron.—Petechial form, patient restless, profoundly prostrated; herpetic and purpuric eruptions, intense aching pains in back and extremities, tongue dry and brown with reddish tip.

TUBERCULOUS MENINGITIS

Tuberculous meningitis is the commonest form of meningitis encountered during infancy. It results from a general tubercular infection in which miliary tubercles develop in the meninges of the brain. In the majority of instances there is a primary focus in the lungs or bronchial glands whence tubercle bacilli

gain entrance into the blood stream and give rise to an acute tubercular process in the brain. It is rarely a primary condition although in many cases the primary focus in some other part of the body is difficult to demonstrate (lungs, bronchial glands, mesenteric glands, cervical glands). At times the meningeal involvement is only part of a general acute miliary tuberculous. Tuberculous meningitis is indicative of a low degree of resistance to tubercular infection as the age incidence indicates. It is perhaps always associated with a recent infection with tuberculosis since patients suffering with chronic tuberculosis rarely develop tuberculous meningitis.

The majority of cases occur from the second to the seventh year, although it may be encountered during the first year of life. The child may show previous signs of a tubercular infection or the disease may develop in an apparently healthy, robust infant. Measles and whooping-cough may act as predisposing causes, especially in children suffering with tuberculous adenitis (cervical or bronchial). It is not uncommon to see it develop in successive children in the same family.

Pathology.—The clinical manifestations of tuberculous meningitis result from the marked serous exudate which develops in conjunction with the meningitis (*"acute internal hydrocephalus"*) and the deposit of tubercles and fibrinous exudate at the base of the brain (*"acute basilar meningitis"*).

The pathological changes found in the brain are miliary tubercles situated along the course of the blood-vessels at the base of the brain, chiefly following the sylvian artery; inflammatory reaction in the pia mater with lymphocytic infiltration; exudation into the ventricles, and more or less infiltration of the brain substance (meningoencephalitis). The blood-vessels are injected and bathed in a serogelatinous exudate. As a result of the intra-ventricular pressure the convolutions may appear flattened. Ordinarily the eruption of tubercles is limited to the base, but tuberculous deposits may also occur in atypical localities producing focal symptoms—cerebral tuberculosis.

The clinical manifestations result from the direct pressure of the inflammatory products upon the roots of the cranial nerves as well as from pressure resulting from the increased weight of the brain and the increased intracranial pressure. The sixth nerve is especially liable to be affected in this manner while the "choked disc" of the optic nerve results from the increased intracranial tension.

Symptoms.—The symptoms of tuberculous meningitis are usually divided into three stages in order to describe the typical evolution of the clinical course of the disease. Many atypical cases, however, are encountered and it is therefore not always possible to trace the symptoms in the order given for a classical case. The age of the patient must also be taken into consideration for the characteristic alterations in the rhythm of the pulse and respirations, the obstipation and scaphoid abdomen seen in older children are often absent during infancy. For the purpose of illustrating the clinical course of a type of case the following description is given:

There is usually a *prodromal period* in which a previously well child shows indications of ill health by a change in disposition, loss of appetite, loss of weight, headache and constipation. It loses interest in its games and playmates, may become exceedingly irritable, hyperesthetic to light and noises and there may be a slight fever. Soon distinct symptoms of *brain irritation* set in; these are notably headache and vomiting. The latter symptom if occurring without an indiscretion in diet, is very suggestive. The vomiting is usually of the projectile type and is due to irritation of the meningeal branch of the vagus. Constipation is of the spastic type and the abdomen is flat or retracted. At this stage a slight rigidity of the neck may also be noted. The child grits its teeth during sleep and shows many other signs of cerebral irritation.

Vasomotor disturbances present during this period are alternate flushing and paleness of the face, and the *tache cérébrale*, a broad red line produced by drawing the finger-nail

across the skin of the abdomen, persisting for a few minutes and indicating vasomotor paresis.

With the progress of the meningeal inflammation the signs of increasing *intracranial pressure* make their appearance. Irregular innervation of various muscles supplied by the cranial nerves is a common symptom of this stage. To these manifestations strabismus and twitching of the facial muscles belong most prominently. There is ptosis and the eyes are often fixed in a characteristic vacant stare. The accumulation of fluid in the ventricles is largely responsible for the pressure symptoms in the centres of the oculo-motor nerve. Likewise, the increased weight of the brain causes it to sag down upon the base of the skull and, by direct pressure upon the abducens, set up an inward deviation of the eyes. The deposit of miliary tubercles and inflammatory exudate upon the basal nerve roots tends to produce paralysis in the parts supplied by them. When the vagus and glosso-pharyngeus become finally involved, death results.

Retraction of the head, opisthotonos, rigidity, twitching and automatic movements of extremities are the chief irritative manifestations of tuberculous meningitis. The most characteristic of these is the retraction of the head, which is a strong presumptive sign of basilar meningitis, although it is frequently seen in "cerebral" pneumonia. This symptom may also disappear in the later stage. Kernig's sign and Brudzinski's neck phenomenon can be demonstrated at this stage of the disease. In infants bulging of the fontanel is a prominent symptom.

In infants emaciation is pronounced and progressive. Convulsions may occur, but they are not a constant feature. Likewise, the shrill, piercing cry, "*cri hydrocephalique*," may or may not be heard.

The final stage is that of *paralysis* in which the child becomes stuporous, the pulse is slow and irregular, the respirations are of Cheyne-Stokes type and the pupils are dilated and fail to react to light. Spasticity of the extremities and convulsive

movements are frequently seen. There is inability to swallow and emaciation progresses rapidly. The reflexes are abolished and urine and feces are passed involuntarily.

The average duration of the disease is from two to three weeks; cases of meningitis persisting for four to five weeks or longer are not likely to be tubercular in nature.

The *prognosis* is always unfavorable. A few authentic cases of tuberculous meningitis which have recovered are on record but they are so exceptional that we should not build up false hopes for the curability of this disease.

The *diagnosis* of tuberculous meningitis is based upon its gradual onset, the presence of the tuberculous diathesis or tuberculous family history; and the development during the early period of the disease of the characteristic symptoms, namely constipation, headache, slowing of the pulse, vomiting and drowsiness. In the later period the appearance of cranial nerve paralysis and choked disc makes the diagnosis more certain.

Cerebrospinal meningitis is differentiated by its rapid onset and acute course; *cerebral hyperemia* by its transitory nature and *hydrocephaloid* by its association with diarrheal or other exhausting disease. The diagnosis can be corroborated by the examination of the cerebrospinal fluid, the characteristics of which are described under "Lumbar Puncture." When the tubercle bacillus cannot be demonstrated in the cerebrospinal fluid its presence may be definitely established by inoculating a guinea pig with the same.

LUMBAR PUNCTURE.

Lumbar puncture was introduced into clinical medicine by Quincke as a method of diagnosis in intracranial affections and it has become one of the most valuable methods of clinical diagnosis at our command. Furthermore, it has also opened up a most useful field in the treatment of cerebrospinal affections. Owing to the continuity of the sub-dural space through-

out the entire cerebrospinal nervous system, it is self-evident that a specimen of fluid withdrawn from the lower end of the dural sac is identical in character with the fluid higher up in the spinal canal, and even within the cranium. Clinical experience has proven this fact so reliable that we now place absolute dependence upon the cerebrospinal fluid obtained by lumbar puncture for information concerning spinal and intracranial pathological conditions. Even in the case of nervous syphilis more dependence is placed upon a Wassermann examination of the cerebrospinal fluid than of the blood.

Technique.—The spinal cord proper terminates in the conus at the second lumbar vertebra where it divides into two coarse strands of fibres, which hug the lateral walls of the spinal canal. These bundles constitute the cauda equina and there is ample space between them for the safe introduction of a needle; besides, they are more or less movable and therefore not readily wounded. Therefore, a small trocar introduced between the spines of the third and fourth or fourth and fifth lumbar vertebræ, will enter the dural sac and the cerebrospinal fluid can in this manner be withdrawn easily and safely for purposes of clinical examination.

The best instrument for performing lumbar puncture is the original Quincke needle. An aspirating needle—10 cm. long and 1mm. in diameter—answers in the case of children. The operation must be performed under the strictest asepsis; this applies to the operator's hands, the instrument, and to the skin at the site of puncture.

It is not always easy to locate the different vertebra by attempting to count them from above downward, but if we remember that a line drawn across the back on a level with the crests of the ilia will intersect the fourth lumbar interspace, it is a simple matter to select either this space or the one above it as the site for puncture. We may puncture as high as the second interspace, but there is not only an imaginary, but an actual advantage in selecting the lowest point, for as Sahli

was able to demonstrate, pus and other elements tend to gravitate to the lowest point, and when present in inconsiderable amount, clear fluid may be withdrawn from the second, while a cloudy one may come from the fourth interspace.

The patient is turned on his right side, and the spinal column bowed as much as possible by flexing the legs upon the abdomen and pressing down upon the buttocks, at the same time bending the upper portion of the back by downward pressure upon the shoulders. Care should be exercised not to exert pressure upon the neck, but always upon the shoulders. The spines of the vertebræ now stand out prominently and we are in a position to plunge between them into the canal. When the patient is comatose no anesthetic is required, and when partly conscious ethyl chloride should be used locally. In young children the laminae of the vertebra are horizontally placed and the interspinous ligament is not very firm. For this reason we can pierce directly between the spines and enter at a right angle to the spinal column. In older children the laminae are somewhat overlapping and the interspinous ligament is tough and firm. Here it is best to pursue the course originally recommended by Quinke, namely, place the point of the needle to the lower side of the median line and a little below the interspace; then direct the needle upward and inward, thus avoiding the ligament and at the same time slipping in between the laminae. In a child two years old the dural sac is penetrated when the needle is inserted for a distance of from 2 to 3 cm., in adults it must penetrate 4 to 6 cm. With a little practice we soon learn to recognize when the needle is in the spinal canal; there is no further resistance and the point can be freely moved. The stilet of the trocar is now removed and the first few drops of fluid are allowed to flow out; the remainder is caught in a sterile graduate, in order to estimate the quantity withdrawn. Ten cc. is sufficient for diagnostic purposes, but when the pressure is great we may withdraw as much as fifty cc. A portion of this can be used for making cultures or for

inoculating guinea pigs. The balance is studied microscopically. The chemical examination is also important.

The study of intracranial pressure is interesting, but of little clinical value in pediatric practice. For practical purposes we can estimate this sufficiently by the force with which the fluid flows from the canula. If a manometer be attached to the canula the pressure can be measured in mm. of mercury. The normal pressure in adults in the prone position is 5 to 7.3 mm. Hg.; a pressure above 15 mm. Hg. is indicative of conditions such as meningitis and brain tumor (Sahli).

Under normal conditions the fluid comes from the canula drop by drop. When the pressure is increased the drops come more rapidly and with considerable effusion it will spurt out in a stream. The stream is not steady and is affected by respiration. In tuberculous meningitis the pressure is higher and more fluid is obtained than in other forms of meningitis. In young infants the fontanel offers an additional means of estimating intracranial tension.

The normal cerebrospinal fluid is clear, colorless and limpid. Its specific gravity varies between a little over 1000 to 1008. There is a trace of serum globulin, a copper reducing substance, presumably glucose, and salts.

The copper reducing substance is usually absent in meningitis, either tuberculous or epidemic, while it is present in poliomyelitis.

The admixture of blood may be due to the wounding of a vein and thus spoil the specimen for gross and microscopic study. It may, however, be due to hemorrhage into the cord or ventricles.

In tuberculous meningitis the fluid is clear. If it is kept in a refrigerator for 24 hours a delicate fibrin mesh-work will form due to spontaneous coagulation of the fibrin present. This coagulum should be placed on a slide and dried and fixed, after which it should be stained for tubercle bacilli. By this procedure the tubercle bacillus can frequently be demonstrated.

In epidemic cerebrospinal meningitis the fluid is cloudy from the beginning, becoming more purulent with the progress of the disease. During convalescence and after serum treatment it begins to clear up.

In poliomyelitis there is but a slight turbidity, the so-called "ground glass" appearance being characteristic of this disease.

In hydrocephalus the fluid is clear, although it contains some leucocytes.

The *cellular elements* which are found in the cerebrospinal fluid are of distinct clinical significance. Normally there is a cell count of but a few leucocytes to the cubic millimeter. In tuberculous meningitis there is a moderate increase of cells, mostly lymphocytes while in poliomyelitis there is a somewhat greater increase of the lymphocytes. In cerebrospinal meningitis polynuclear cells are found in great abundance.

French writers lay great stress upon the predominance of lymphocytes in tuberculous meningitis. While the value of cytodiagnosis in cerebral inflammations is not without limitations, still a marked increase in lymphocytes over polynuclear elements is strong presumptive evidence in favor of a tuberculous infection.

In purulent exudates ordinary cover-glass preparations stained with methylene blue are sufficient for the study of the bacteria present. Streptococci and pneumococci are recognized by their morphology while the micrococcus of epidemic cerebrospinal fever is a diplococcus similar in appearance to the gonococcus. The majority of these diplococci will be found within the pus cells. As Park puts it, the cells are crowded with the diplococci.

Noguchi's globulin test is a valuable diagnostic aid in the study of the cerebrospinal fluid. It is present in the early stages of poliomyelitis and strongly positive in all forms of meningitis. It is also positive in nervous syphilis. For the technique of Noguchi's test the reader is referred to any work on clinical diagnosis.

The *indications* for lumbar puncture are any obscure cerebral condition in which there is clinical evidence of inflammation of the meninges or of intracranial pressure. So far its chief value has been that of a diagnostic aid, the importance of which cannot be questioned. Temporary relief of symptoms, and convulsions, has also attended its use, and in the control of uremic convulsions and coma it has proven of value. Complications, such as pneumonia, contraindicate it.

LETHARGIC, OR EPIDEMIC ENCEPHALITIS.

Lethargic encephalitis is an acute infectious disease occurring epidemically which first attracted attention in 1917 when a number of cases were reported from Austria. In 1918 it broke out epidemically both in England and France and since that time it has been observed extensively in America. While children are affected with the disease, still it is more commonly observed in adults and, in this respect, differs from poliomyelitis which is characteristically a disease of childhood.

The micro-organism, or virus causing epidemic encephalitis has not been isolated. Owing to the resemblance of the symptoms to those encountered in botulism it was at first believed that the condition was the result of the poor food in the countries directly affected by the World War. However, subsequent developments have definitely ruled out this factor as an etiological one. A relationship with influenza was also considered in the etiology of encephalitis because the first epidemic followed closely in the wake of the great influenza epidemic.

Pathological studies of fatal cases have shown perivascular lymphocytic infiltration chiefly affecting the upper portion of the pons and the basal nuclei. These changes explain the presence of the ophthalmoplegia and facial nerve involvement. There was no degeneration of ganglion cells as occurs in poliomyelitis. Many of the general manifestations of the disease must be explained on the basis of the associated toxemia.

Symptoms.—The leading clinical manifestations of lethar-

gic encephalitis are drowsiness and stupor, whence the disease gets its name, associated with general muscular weakness and involvement of the eye muscles. While in typical cases third nerve and facial nerve paralysis are striking symptoms, still many atypical and incomplete forms are encountered as in the case of poliomyelitis, making the diagnosis difficult at times. When the characteristic focal lesions are present, ophthalmoplegia or facial paralysis or both will develop. When, however, the pathological changes are not pronounced the general toxic features of the disease alone may be present. McNulty describes the following clinical types of epidemic encephalitis:

1. Cases with general manifestations but without paralysis.
2. Cases with third nerve paralysis.
3. Cases with facial paralysis.
4. Cases with spinal manifestations.
5. Cases with peripheral nerve involvement.
6. Abortive cases.

The earliest cases of lethargic encephalitis occurring in children were reported by Batten and Still (*Lancet*, May, 1918), who called the condition *epidemic stupor in children*. The chief symptoms were stupor from which the patient could be roused; muscular rigidity and tremor; mask-like expression of the face; nystagmus and inco-ordination of the eye muscles. There was no retraction of the head or disturbance of the reflexes. The cerebrospinal fluid showed nothing abnormal and the disease tended to recovery after the children had lain in the condition of stupor for several weeks. Netter reported a similar epidemic occurring in the children of Paris and Neal (*Archives of Pediatrics*, June, 1920) has contributed an exhaustive study of the cases occurring in New York City.

Lethargy is the most striking and most constant symptom being encountered in the majority of cases. The patient is not completely unconscious and can be roused and often will answer questions intelligently but in a slow, hesitating manner. There is moderate fever in the beginning but this only lasts for about a week. Convulsions are rare. The cerebrospinal fluid is increased but it is clear and there is only a slight increase of

globulin and cellular elements. In this respect it differs from the fluid obtained in cases of poliomyelitis and meningitis.

A peculiar type of encephalitis in children in which insomnia and mental disturbances dominate the clinical picture has recently been reported by Happ and Blackfan. Similar cases have been observed by other pediatricists since the above article appeared.

The duration is protracted in the majority of cases, the symptoms remaining stationary after their full development, for a period of several weeks and then gradually subsiding. Mild cases will run their course in from three to four weeks while the more severe ones may last as many months. The mortality is about 20 per cent, being lower than in the meningeal type of poliomyelitis. Death is usually due to bulbar paralysis.

Diagnosis.—The gradual onset of the symptoms, namely, mental irritability followed by drowsiness and lethargy associated with the development of an ophthalmoplegia should suggest encephalitis although tuberculous meningitis may present a similar clinical picture in its early stages. Lumbar puncture should always be performed both for its diagnostic as well as therapeutic value. The protracted course of the disease soon excludes tuberculous meningitis, and the absence of vomiting in the beginning of the disease speaks against meningitis. Poliomyelitis is more abrupt in its onset, paralysis and signs of meningeal irritation are present as well as in meningitis (Kernig's sign; rigidity of the neck; exaggerated reflexes) while these symptoms are absent in encephalitis.

Treatment.—The chief remedy for epidemic encephalitis is *gelsemium*. The symptoms upon which it is indicated are drowsiness; stupidity; tremulous weakness; extreme prostration and muscular weakness; occipital headache, moderate fever; paralysis of the oculo-motor nerve.

In the cases characterized by insomnia and mental symptoms *belladonna* is indicated. If the symptoms are not controlled by *belladonna*, *hyoscyamus* should be prescribed. For the late

manifestations and residual paralysis *causticum* is the most important remedy. Indications for other remedies which may be required in special cases will be found under the treatment of meningitis. *Lumbar puncture* seems to exert a beneficial influence over the course of the disease and should be systematically performed at intervals of a week or less, according to indications.

HYDROCEPHALUS.

Hydrocephalus is a chronic idiopathic disease, in which there is an excess of cerebrospinal fluid in the cranial cavity (*Hydrops cerebri*). The so-called acute hydrocephalus is a synonym for tuberculous meningitis and has no relationship to chronic hydrocephalus.

There are two forms of the disease, namely, *external hydrocephalus* and *internal hydrocephalus*. In the former there is a subdural accumulation of fluid, between the dura mater and the arachnoid. This is a rare form, almost invariably occurring secondarily to a congenital defect of the brain, meningeal hemorrhage, pachymeningitis, or atrophy of the brain. In the last instance the serous effusion occupies the space left vacant by the deficient brain, and it is spoken of as *hydrocephalus ex vacuo*.

Chronic internal hydrocephalus is the commonest form of the disease, and the one usually referred to in speaking of hydrocephalus. The largest number of cases are congenital. The head may be so large at full term as to impede delivery, or the effusion be but trifling and accumulate so slowly that the head does not become noticeably enlarged until several weeks after birth. A slight degree of enlargement of the head is noted in most cases at birth but in some instances it may be so slight that it escapes notice and the condition is not recognized clinically until the child is several months old.

Internal hydrocephalus is almost invariably a primary condition. In rare instances it is found associated with tumors of inflammatory processes at the base of the brain when such con-

ditions cause obstruction of the foramen of Magendie or obliterate the communications between the ventricles of the brain. Accumulation of cerebrospinal fluid in the ventricles may result from diminished resistance of the cranial walls, and also from causes directly increasing the blood-pressure in the brain, i. e., whooping-cough, bronchitis, emphysema and convulsions. Such conditions however are transient and rarely lead to a permanent hydrocephalus. The head is enlarged in rickets, owing to the abnormal softness of the cranial bones and the malnutrition but here again true hydrocephalus is rare. In congenital syphilis, however, a moderate degree of hydrocephalus is one of the characteristic clinical manifestations of the disease. Under specific treatment improvement usually occurs which unfortunately does not take place in idiopathic hydrocephalus.

The brain is greatly distended, the convolutions become obliterated and the cortex may become a mere shell or the brain appear as a large cyst. The cranial bones are thin and the sutures widely separated; supernumerary bones are commonly found. In rare instances premature ossification of the cranium occurs with hydrocephalus. Spina bifida and other congenital defects may be found associated. The amount of fluid varies from several pints to more than a gallon. It resembles the normal cerebrospinal fluid.

The lateral ventricles show the greatest amount of distention. In about half of the cases the communication with the spinal subdural space is obliterated and a lumbar puncture will prove negative when this is the case. The third and fourth ventricles likewise will either be found normal or distended according to whether the choroid plexuses are normal or sufficiently enlarged to occlude the ventricular aqueducts.

Symptoms.—The head is rounded, its size much out of proportion to the rest of the body and in its relation to the development of the face, and the fontanels and sutures are wide open and tense. In external hydrocephalus the enlarge-

ment is usually not so pronounced, and when the skull is still soft fluctuation may be elicited over the head.

It is necessary to remember the normal circumference of the head at different periods of infancy in order to determine whether the head be abnormally large. At birth this is about fourteen inches, and at the end of the first year nineteen inches. Beside this, the relationship of the circumference of the head to the chest is important to bear in mind, the circumference of the head at birth exceeding that of the chest by half an inch; later, during the entire period of infancy, the two measurements are practically equal. From these data it is easy to determine an abnormally developing head. Every cranial enlargement does not, however, indicate hydrocephalus, the most important condition to be differentiated being rickets. Hydrocephalus may occur *without enlargement* of the head. In such cases there is either premature ossification of the skull or a late onset of the disease. They are generally idiots and die early (Holt). It is impossible to recognize this condition during life.

The rate at which the head enlarges varies greatly; the earlier and more rapidly the enlargement develops the more serious is the prognosis. Cerebral symptoms are slight, often entirely wanting. The development of the child is, however, much retarded, and the majority of cases die early of marasmus. Those surviving this period die in early childhood, as a rule, from some intercurrent disease. The mind becomes affected and many are idiots. They are irritable and often show evidence of violent temper. In others, again, the intelligence is but slightly interfered with and they may live even to reach adult life, being, however, both physically and mentally retarded in development and helpless on account of the great weight of their head. A moderate degree of hydrocephalus is not incompatible with normal mental development. Some cases evidently become arrested and grow up as normal individuals with an abnormally large head of the characteristic shape and show average intelligence.

The *differential diagnosis* between hydrocephalous and *rickets* should present no difficulties. The hydrocephalic head is enlarged out of all proportion to the rest of the body and presents a regular rounded outline. The eyes are deflected downward, so that the lower lid crosses the iris higher than normal. The outline of the skull is globular and the forehead is prominent and bulging. The face is small in comparison with the head. The fontanel bulge and pulsate, and the sutures are widely separated, while the cranial bones feel thin. The veins of the scalp are prominent and distended, owing to interference with the return circulation of the brain. Nystagmus and strabismus are frequently noted. Optic nerve atrophy is also noted in some cases as a late development. In *rickets*, on the other hand, the head is square and the centres of ossification in the frontal and parietal bones are hypertrophied. The skull is hard excepting in the occipital region, where craniotabes may be present. Enlarged epiphyses and deformities in the extremities are associated.

The *treatment* is unsatisfactory. Up to the present time no form of either medical or surgical treatment has proven itself to be of any distinct benefit. *Mercury* and *potassium iodide* should be used in all cases seen early and a certain percentage of cases will be benefited by these remedies but every case is not specific and does not, therefore, respond to this treatment.

Bartlett (Goodno's *Practice*) mentions some cures effected by the application of solar heat, which is a harmless measure that may at least be tried. "The method consists in exposing the child's occiput to the direct rays of the sun for twenty minutes each day, gradually increasing the duration of the seance until the limit of thirty or forty minutes is reached. It is believed that the local sweating acts to remove a portion of the effusion, while the thermic heat aids nutrition." Strapping the head with adhesive plaster; repeated tappings of the lateral ventricles; tappings followed by the injection of iodine; draining the ventricles into the areolar tissue of the scalp are some of

the surgical methods which have been tried and are advocated by some surgeons.

CONVULSIVE AFFECTIONS.

Eclampsia, or *Infantile Convulsions*.—General convulsions are of common occurrence in infants of the spasmophilic diathesis (see “Spasmophilia”). Apparently insignificant causes like teething can precipitate a convulsion in such infants. Rickets also predisposes to convulsions. Infants and children in whom there is no evidence of spasmophilia may have convulsions from conditions which are, however, of a more serious nature. Thus, acute infectious diseases are frequently ushered in with convulsions. In meningitis, encephalitis, hydrocephalus and brain tumor, convulsions occur symptomatically. Idiopathic convulsions in older children are usually epileptic in nature. Convulsions occurring in the newborn are frequently due to cortical irritation from a birth injury. The status lymphaticus may be the cause of a fatal convulsion.

The anatomical lesions found in children dying in convulsions are by no means constant or characteristic. The changes occurring in the brain are probably anemic, followed by venous hyperemia. When intense congestion, serous effusion and punctate hemorrhages are found after death they are to be looked upon as a result of the convulsion and not as a cause of the same, death having resulted from asphyxia. The initial stage of a meningitis may also be found; or, if the convulsion depends upon organic brain disease, such a condition becomes evident. In some instances the anatomical findings of the status lymphaticus are present.

Symptoms.—Infantile convulsions are most frequently general although a localized or partial convulsion may result as well from reflex irritation as from organic disease. In such cases the subsequent course of the disease must be followed before a final diagnosis can be made. True local convulsions or *Jacksonian epilepsy*, repeatedly commence in one extremity

and if they do not remain local, at least continue so for an appreciable time (Herter). This form of convulsion is indicative of organic disease, the nervous discharge commencing at the seat of irritation. With it there is no loss of consciousness. Prodromal symptoms are therefore usually present, indicating the commencement of a general convulsion. They may be so slight as to be entirely overlooked, or they may manifest themselves as extreme restlessness, twitching of the mouth, eyelids, extremities, and rolling of the eyes.

The convulsion proper is very similar to an epileptic fit. The child becomes suddenly rigid, the head being thrown back, the hands clenched, with thumbs buried in the palms, and the extremities stiffen out. This stage is only of short duration, not as long as in a true epileptic attack, while the succeeding stage, consisting of intermittent spasmodic contractions of the extremities, is comparatively longer. During this stage the entire body is seen to take part in alternate rhythmical contraction and relaxation. The child is unconscious, and may involuntarily pass both urine and feces. In the course of a few minutes to half an hour, according to the gravity of the case, the spasms gradually subside, leaving the child in a soporous condition. It is not uncommon for several convulsions to occur in succession, as repeated convulsive seizures create a susceptibility from which the nervous system recovers itself with difficulty.

Prognosis.—This depends upon the nature of the exciting cause and the course pursued by the seizure. When convulsions recur in rapid succession, or when associated with laryngismus stridulus, the prognosis becomes grave. Likewise in convulsions occurring with uremia or with meningitis, extensive hemorrhage, or other serious intra-cranial lesion, the prognosis is grave. Should the convulsive habit become firmly established, there is danger of epilepsy developing. When the convulsions are unmistakably due to spasmophilia the prognosis is good as this condition is amenable to treatment.

Diagnosis.—The differentiation of symptomatic from idiopathic eclampsia rests upon a proper examination of the patient for evidence of disease elsewhere. Thus, with convulsions ushering in the infectious fevers, there are always the symptoms belonging to the stage of invasion of the particular fever in question. Convulsions are quite common with the onset of pneumonia, especially apex pneumonia. In uremic convulsions the urine establishes the diagnosis. Those due to reflex irritation give evidence of such a source of irritation, and purely spasmophilic and rachitic cases show evidence of these conditions. Intra-cranial disturbances are recognized by characteristic neurological findings which can be demonstrated both before and after the convulsions have occurred.

Convulsions occurring shortly after birth are usually due to meningeal hemorrhage. Unilateral spasms may occur from cortical hemorrhage, as a result of whooping-cough, trauma of idiopathic origin.

Epilepsy is to be suspected when repeated convulsive seizures occur in children over three years of age, notwithstanding the absence of any source of reflex irritation or a toxic cause for the attack. Other symptoms, such as an aura and stigmata of degeneration, are usually ascertainable.

Treatment.—All exciting causes must be removed at once whenever this is possible, and the predisposing cause is to be overcome by attending to the child's general condition. Constitutional and dietetic treatment together with an abundance of fresh air and sunshine, are indispensable here. (See *Rickets* and *Spasmophilia*.)

As gastro-intestinal irritation plays such an important role in the precipitation of convulsive seizures, the bowels should at once be emptied when the attack is suspected to arise from this source.

During the seizure every article of clothing should be loosened. If the convulsion lasts for any considerable length of time a warm bath, together with cold applications to the head,

is indicated. In long-continued or recurring convulsions a hot pack may be resorted to.

The most frequently indicated remedies are *Belladonna*, *cuprum*, *ignatia* and *magnesia phos* basing our prescription purely upon the occurrence of convulsions. But when the convulsion is symptomatic, the results of treatment will be more satisfactory if we direct our attention to the exciting cause instead of looking upon the convulsion as an independent disease. In some instances it becomes necessary to check the convulsions when they recur at dangerously frequent intervals by giving a rectal dose of *chloral hydrate* or *potassium bromide*.

Bell.—Convulsions, with flushed face; dilated pupils; cerebral congestion; throbbing carotids; pyrexia. Indicated in those cases ushering in the infectious fevers, in some reflex convulsions, and in convulsions occurring in the early stages of meningitis.

Cuprum.—Convulsions beginning in the fingers and toes, becoming general, with marked cyanosis. Spasm of the glottis is associated with these cases. Convulsions occurring during the eruptive fever when the rash disappears; whooping-cough; meningitis. *Cuprum ars.* is more valuable in uremic convulsions.

Cina.—Reflex convulsions from irritation of the intestinal tract, whether due to worms or not. The spasmodic movements are often confined to the eyes and face, continued with irregular jerkings of the extremities. In this respect it is similar to *chamomilla*, which presents many of the premonitory symptoms of eclampsia, the child being feverish, irritable, and suffering with intestinal colic or painful teething. In such cases *chamomilla* will frequently ward off a convulsion.

Cicuta.—The convulsion comes on suddenly without premonitory signs. The stage of tonic spasm is well marked, and the child may remain rigid for a long time, only a few jerks of the extremities being noticed during the attack. *Cicuta* is especially indicated in convulsions occurring in cases of cerebrospinal meningitis.

Ignatia.—Convulsions in nervous subjects brought on by fright or peripheral irritation. The vascular excitement of *belladonna* is not present in these cases, and the face is pale instead of hot and flushed, as in the latter remedy. Neurotic, hysterical children.

Magnesia phos.—Idiopathic convulsions; defective nutrition of the nervous system; spasmophilia. (See *Epilepsy*.)

Opium.—Convulsions in cerebral hemorrhage. There is trembling of the whole body; purplish color of face; stertorous breathing and sopor; post-epileptic stupor.

EPILEPSY.

Idiopathic epilepsy is a chronic disease characterized by the occurrence of attacks of unconsciousness with general convulsions (*grand mal*) or by recurring attacks of momentary loss of consciousness alone (*petit mal*).

Etiology.—Heredity plays a prominent role in the etiology of epilepsy. The family history may reveal the existence of epilepsy, insanity or some other serious nervous affection in the parents or near blood relations. Parental syphilis and alcoholism are also looked upon as etiological factors. In many instances a history of convulsions in infancy and early childhood is obtained.

Many epileptics show the stigmata of degeneration to a marked degree. They may be vicious and criminally inclined or are intellectually and morally defective. Startling exceptions in the form of geniuses, are observed. As to age, the period of puberty furnishes the majority of cases. It only rarely develops before the third year. Sachs (*The Nervous Diseases of Children*) is of the opinion that *hereditary* (idiopathic) epilepsy is not as common as is generally supposed, many cases being accepted as such because a former cerebral lesion or a traumatism to the head has been overlooked owing to the disappearance of the paralysis and other symptoms due to such a lesion, from which, however, the epilepsy springs. To this category belong

those cases of epilepsy associated with infantile cerebral palsies and defective development of the brain.

The *exciting cause* of the seizure is most often found in disturbances of the digestive tract. Overeating is a common factor. Acute indigestion, either through reflex irritation or auto-intoxication, will frequently precipitate an attack. Reflex irritation from phimosis, eyestrain, worms, etc., exerts a similar influence. Emotional excitement, excessive physical exertion, and poorly ventilated or crowded apartments are most disadvantageous to the epileptic. In several of my cases the first seizure developed after a slight traumatism, the psychic effect no doubt being more to blame than the accident itself.

A constant pathological lesion is not found. Judging from our knowledge of the physiology of the brain and the symptoms produced by irritation and organic disease of the cortex in the Rolandic area, it is reasonable to suppose that the pathologic condition is located here. Indeed, a number of observers, notably Van Giesen and Bleuler, have demonstrated changes in the cortical cells and in the neuroglia. Lesions in the basal ganglia have also been described. These are probably in the nature of secondary changes. Many cases show evidences of the status lymphaticus.

Symptoms.—An attack of *petit mal* is characterized by a momentary loss of consciousness, unaccompanied by convulsions or other nervous phenomena. In children it is often looked upon as mere absent-mindedness or a fainting spell; in older subjects it is more likely to be confounded with vertigo, with which, it is unnecessary to say, it has nothing in common. After this condition has once been fully established, a change in the child's mentality becomes manifest; it may go over into the convulsive form or exist in conjunction with the same.

Besides *petit mal* there are numerous other forms of incomplete seizure, all, however, attended by *momentary loss of consciousness*. There may be merely twitching of certain muscles, notably in the arm and face; a sudden impulse to run

forward or perform other automatic movements, of which the patient is unconscious. Sometimes *coma* exists without convulsions, or the child may have auræ for a long time before the convulsions make their appearance. There are also certain *psychic equivalents* of the epileptic seizure, in the form of maniacal and other insane acts. Following the fit, the patient may for several days perform acts for which he is irresponsible.

An attack of *grand mal* is very similar to an attack of infantile convulsions. Other symptoms, however, which are not found with ordinary convulsions are present and the various stages are more sharply defined and characteristic. The following stages are to be observed:

(1) The *aura*, or *prodromal stage*. This usually consists of a sensory disturbance, which may be variously described as a tingling; feeling of numbness; crawling; sensation of a gust of wind directed upon the affected part; hallucinations of sight, smell and hearing. There may also be motor disturbances, and the character of the aura will in many instances point to involvement of a special area of the cerebral cortex.

(2) The *initial cry*. This marks the commencement of the stage of tonic spasm. The patient utters a loud cry, as a result of the spasmodic contraction of the respiratory muscles forcing the air through the partially closed glottis, whereupon he loses consciousness and falls to the ground.

(3) The *tonic spasm*. During this stage the body is perfectly rigid, the legs extended, the arms flexed and the hands clenched, the thumbs being pressed into the palms of the hands. The head may be retracted, as in *opisthotonus* (young children), or it may be drawn to one side, the eyes being fixed and pointing in the same direction. The *pupils* are immovably dilated. The face, at first pale, now becomes reddened, and even cyanotic, if this stage is prolonged. The jaws are set, and the tongue is frequently caught between the teeth. The stage of tonic spasm lasts for a period of about a minute, at the end of which time it gradually subsides, being followed by—

(4) The stage of *clonic spasm*. This consists of alternate relaxation and contraction of the muscles of the extremities and thorax, persisting for several minutes (seldom over five minutes). Through these movements the body is thrown into violent action, and frothy saliva is ejected from the mouth, the tongue quite frequently being caught between the teeth and badly bitten. Urine and feces are frequently passed involuntarily. The movements gradually subside and the patient goes over into—

(5) The stage of *stupor*. Post-epileptic stupor is a profound sleep from which the patient may be temporarily aroused, but soon relapses into unconsciousness. This may last for several hours. The pupils are dilated.

Prognosis.—It is the consensus of opinion among neurologists that true epilepsy is an incurable disease. Some cases of a mild type which have received early care and treatment may recover. Spontaneous recovery also may occur in rare instances and cures through surgery have been reported although the failures are too frequent to give surgery much credit. The best results that we can expect from treatment are a reduction in the number of seizures and the general improvement of the patient's physical and mental condition.

Diagnosis.—*Eclampsia*: Prior to the age of three years; the convulsions are of longer duration and signs of rickets or spasmophilia can be demonstrated. Eclampsia responds to dietetic and constitutional treatment while this has little effect on true epilepsy.

Hysteriod convulsions are usually precipitated by emotional excitement; rigidity is marked, followed by irregular movements of the extremities; the duration is much longer than an epileptic seizure, and there is no biting of the tongue or involuntary micturition and defecation (Gowers).

Other conditions to be thought of are *uremic* and other *toxic convulsions*, and, in the case of *petit mal*, *syncope* and *vertigo* must be excluded.

We may be called upon to make a diagnosis in a case where

the occurrence of convulsions is not known. Thus, we may find a patient in post-epileptic coma, or have to deal with a case of nocturnal epilepsy where the convulsions have taken place unobserved. Post-epileptic coma is distinguished from uremia by the presence of dilated pupils and the absence of albuminuria and casts in sufficient amount to indicate nephritis. The tongue should be carefully examined for scars. Strumpell lays stress upon a careful inspection of the conjunctivæ and face for punctate hemorrhages. When these are seen in a patient who awakens in the morning dull and confused we have strong presumptive evidences of nocturnal epilepsy. When this is associated with enuresis the presumption is still stronger. Besides, in forming an estimate of the true nature of any condition associated with disturbed or temporary loss of consciousness the family history, the child's mental development and the presence of stigmata of degeneration play an important role.

Treatment.—All sources of reflex irritation, such as phimosis, cicatrices, errors of refraction and nasal defects must be corrected. The diet is of great importance. The patient should be kept mainly on a vegetable diet, allowing milk regularly, and poultry and fish occasionally; furthermore, the stomach must never be overloaded, and, besides prohibiting meat, all indigestible articles of food, such as pastry, rich desserts, etc., must be strictly avoided (Bartlett).

In cases of malnutrition, meat may occasionally be allowed, but a liberal meat diet is always injurious to the epileptic. Cases in which convulsions had ceased under an exclusive vegetables and milk diet invariably relapsed when meat was allowed, no change in the medicinal treatment having been made (Thompson, *Practical Dietetics*). Cod liver oil is indicated in the rachitic and strumous.

It is a noteworthy fact that an excess of *indican* is found in the urine of many epileptics just about the time of the seizure (Herter), being formed in the intestines from the excessive

putrefaction of proteids. This points to the necessity of preventing intestinal putrefaction, which may be at least partially accomplished by careful regulation of the diet. Indicanuria is produced not only by the indigestion of albuminous food, but also as a result of muscular atony of the stomach and sub-acidity. As the lactic acid bacillus is antagonistic to the colon bacillus, and should in fact predominate over the latter in the small intestines in childhood, it is readily seen why a milk diet and the prohibiting of meat is so beneficial in epilepsy.

Excessive physical exertion must be avoided, while judicious out-of-door exercise proves of the greatest benefit.

During an attack the patient should be protected from injuring himself. A towel or other available article may be inserted between the teeth to prevent biting the tongue, and the clothing should immediately be loosened. The inhalation of *amyl nitrite* sometimes shortens the attack.

The best therapeutic results are obtained from remedies selected upon general indications, taking into consideration the patient's mental, temperamental and diathetic peculiarities; also, any disturbances in the alimentary, respiratory, genito-urinary tract, etc. For this reason such remedies as *cicuta*, *hydrocyanic acid*, *œnanthe crocata* and *solanum* are rarely of positive value. On the other hand, *argentum nitr.*, *calc. carb.* and *phos.*, *lycop.*, *nux vomica*, *pulsatilla*, *silica* and *sulphur* are of service. *Magnesia phos.* 3x trit. has proven clinically useful in a number of cases of epilepsy.

Where disorders of the digestive tract and lithemic symptoms are prominent conditions *nux vom.*, *lycopodium*, *cina* and *sepia* stand out prominently.

In *petit mal* I have obtained good results from *cannabis indica* in small doses. The seizures occurred less frequently and the child's general condition was improved. Another remedy from which I have obtained results and practically use as a routine in beginning the treatment of any case is *santonin*. Whether or not worms are present, *santonin* certainly is help-

ful in many cases of intestinal toxemia and its empiric use in epilepsy is frequently followed by good results.

The following résumé is given in order to call attention to the guiding indications for the important remedies:

Arnica.—Recent traumatic cases.

Arg. nitr.—Old-looking face, pupils dilated before paroxysm for a day or two, flatulent dyspepsia with cardiac palpitation, apprehensiveness and depression of spirits, attacks of hemi-crania, periodic trembling of body and paralytic weakness, epilepsy from fright, masturbation, menstrual difficulties.

Arsen.—Anemic, weakly subjects. Burning in the spine, burning in the stomach and bowels after eating, diarrhea with smarting about anus. *Petit mal*.

Bell.—Violent convulsions, with marked cerebral congestions; mania. Prodromal symptoms consist of flushing of the face, throbbing of the carotids; wild, staring expression; feeling of suffocation. During the interval, throbbing headache, vertigo, flushing of the face with burning heat, easily frightened, night terrors, enuresis. *Stramonium* is similar in many respects. Symptoms brought on by fright, with great nervous excitement; spasmodic constriction of the throat, gyratory movements of extremities and threatened convulsions. *Stramonium* is frequently of service when *belladonna* has failed to give relief, or its chances for doing good have slipped by, as it is of no service in old cases. The cases in which *hyoscyamus* has proven so beneficial are undoubtedly hysterical in nature, as Jahr intimates. Such causes as "disappointed love, jealousy, and grief," mentioned under the etiology of *hyoscyamus*, point to the hysterical element in these attacks, as also such symptoms as "attempts at swallowing fluids renew the attacks," and "inclined to talk a great deal after the attacks; slight wandering of the mind."

Bufo.—Bojanus (*Die Hom. Therapeutik in ihrer Anwendung auf die Operat. Chirurg.*, 1880) reported a series of twenty-two cured cases of epilepsy, among which four were

cured by the use of *bufo* alone, three with *bufo* followed by *salamander*, and two with *bufo* in conjunction with *lachesis* and *ignatia*. He gives no special indications for this remedy. "After fright or onanism; attacks at night, followed by some hours of coma; loss of consciousness and falling down; tonic and clonic spasms; turgescence and distortion of face; bites tongue; involuntary emission of urine; the lower extremities are more in motion than the upper ones."— (C. G. R.)

Calc carb.—Scrofulous diathesis and leucophlegmatic temperament. Anemia; catarrhal and cutaneous affections; prominent belly; cold hands, sweaty feet; sweating about the head. "Frequently indicated after *sulphur*," or in conjunction with *belladonna*.

Cannabis Indica.—Clinically useful in petit mal. Allen (*Handbook of Materia Medica*) gives the following symptoms: Absent-minded, *forgetful* of what he intended to write or speak so that he cannot finish a sentence; forgetful of his last words and ideas. *Unconsciousness every few minutes*. Misapprehensions concerning time and space.

Causticum.—Where the mind is affected and paralytic affections are associated with the epilepsy. Degenerative changes in the nervous system. Paralytic weakness after the seizure is marked.

Cicuta.—Violent epileptiform spasms, accompanied by puffed, bluish face; fixed, staring eyes; terminating in trembling and long-continued sopor. Intestinal irritation, with venous congestion of abdomen.

Cimicifuga rac.—Epilepsy associated with disturbances in the female generative organs.

Cina and *santonin* are useful in most cases. Ridding the intestinal tract of parasites is one of the first things to be thought of in epilepsy in children. Besides the symptoms directly referable to worms there are a number of others calling for *cina*, particularly those referable to the disposition, the appetite and general nutrition.

Cuprum.—Clear, idiopathic cases without organic lesions. The attacks may have been precipitated by fright, mental excitement, or suppressed exanthemata. Nocturnal epilepsy. The attack is typical, and cyanosis is usually marked.

Gels.—Dull occipital headache before attack; languor; drooping of eyelids; easily frightened into diarrhea; prolonged spasm of the glottis during attack.

Ignatia.—This remedy is especially suited to ordinary cases of epilepsy in children. They are exceedingly nervous and easily frightened, irritable and peevish, and difficult to control. Jahr considered it the most valuable remedy with which to begin a case.

Indigo.—Depression of spirits. Excitable, furious and easily angered before the attack. Melancholy, timid or gloomy after the attack (L. M. Kenyon). From the isopathic standpoint, *indican* in homeopathic doses might prove useful.

Nux vom.—Indigestion with attacks of canine hunger; constipation, tongue coated posteriorly, bad taste, headache on rising in morning, with irritability (*lycop*., great irritability after sleep) and anorexia, especially mornings. *Nux vom*. and *lycop*., are very important general remedies for the epileptic.

Opium.—Prolonged post-epileptic stupor. Nocturnal cases, with mental derangements.

Silica.—Lack of animal heat; strumous and rachitic diathesis; neurasthenia; pale, transparent skin; profuse sweat after the seizure. "Epilepsy, the aura begins in the solar plexus. Chronic effects of fright and nervous shock. Great irritability; constant restlessness" (T. F. Allen).

Sulphur.—Scrofulous or exudative diathesis. It is unnecessary to describe the characteristic *sulphur* child here. *Sulphur* is also important as an intercurrent, or in cases not responding to the usual list of remedies.

Bromides.—The administration of *bromides* in epilepsy should not be a routine procedure but should only be resorted to when other measures fail to keep the number of seizures within safe bounds.

"It is certain that very few cases have been permanently cured by the administration of *bromides*; but unquestionably they serve an admirable purpose in checking the number of attacks and in diminishing their severity. To accomplish this end the *bromide salts* should be administered according to a definite plan. It has been my practice to give preference to the *bromide of sodium*, which I employ, according to the age of the patient, in ten or fifteen-grain doses, three times a day. If given in a wineglassful of (alkaline) water after meals the gastric functions will not be seriously impaired. . . . In the case of nocturnal attacks the medicine should be given before going to bed (the entire daily dose), and at no other time."—(Sachs, *Nervous Diseases of Children*.) The method recommended by Seguin (*New York Med. Jour.*, March, 1890) has many followers. It consists in the administration of the larger part of the full daily dose shortly before the time when a seizure is to be expected. During the interval a much smaller dose is employed, and the *bromide* is always given highly diluted.

According to Bayley, the *bromide of strontium* is less irritating, produces less acne and has seemed to him more satisfactory in results than those obtained from the *sodium* or *potassium salt*. He gives from ten to sixty drops of a saturated solution (each drop representing about $\frac{1}{2}$ grain of the salt) after meals, well diluted. If favorable effect is noted, sufficient dosage is maintained to stave off the paroxysms. It has been claimed recently that the action of the *bromides* is augmented and that therefore the dose can be reduced if we entirely interdict the use of table salt at the time the patient is taking *bromides*.

As soon as the paroxysms are controlled the dose is decreased to a minimum, but the remedy should not be withdrawn immediately.

CHOREA.

Chorea, or St. Vitus' dance, is one of the commonest nervous diseases of childhood. It is a neurosis characterized by the presence of irregular, purposeless, involuntary muscular contractions in various parts of the body, usually of wide distribution, and associated with a loss of muscular tone and disturbed co-ordination of voluntary movements. The onset is acute and the course pursues a sub-acute character. The relationship of chorea to rheumatism is one of its most noteworthy features.

Etiology.—There are evidently two classes of chorea. In the one we can find no evidence of rheumatism or endocarditis. This form is encountered in delicate and neurotic children and usually develops as a result of insufficient fresh air, rest and proper diet in conjunction with the strain of school life. The first symptoms of chorea may develop after a fright or some emotional shock. For this reason we see so many cases developing in the spring of the year; in other words, towards the close of the school term. In every children's clinic the large number of pale, thin, ambitious children, mainly girls, that come regularly with symptoms of chorea in March and April stands in distinct contrast to the scarcity of these cases in the fall.

Hodge has shown that as a result of fatigue the nerve cells shrink in size, their nuclei and nucleoli become shrivelled and the lenticular granules of the protoplasm, probably nutrient, disappear. While under ordinary conditions the cell is promptly restored to normal after a period of rest, a much longer time, and sometimes a protracted period of rest, is required for this restoration in anemic, neurotic children.

Griesbach's interesting experiments with the esthesiometer have given valuable data in the study of school-fatigue in children. When this method of investigation shows that recuperation is sub-normal in a given case we should accept this as a danger-signal, for if fatigue is prolonged it becomes cumu-

lative and then complete recuperation is impossible so long as the child is kept at school (La Fetra).

In the other class of cases the etiological factor is distinctly a rheumatic infection. Rheumatic manifestations (arthritis and endocarditis) may precede, co-exist with or follow the choreic attack.

Streptococci have been isolated from the blood and nervous system in a few fatal cases of chorea by Westphal and by Wassermann; tonsilitis has also been observed to precede attacks of chorea, as in rheumatism.

The opponents of the rheumatic theory of the etiology of chorea have been misled to a certain extent by a failure to understand the clinical course pursued by rheumatism in the child. If we remember that rheumatic infection does not necessarily mean polyarthritis, but that certain forms of sore throat; vague joint pains or pains in the muscles and tendons accompanied by fever; growing pains and primary endocarditis itself are all manifestations of a rheumatic infection in childhood we will concede a much higher percentage of rheumatism in our choreic patients than otherwise. Heubner, makes the rather sweeping statement that chorea is the commonest form of rheumatism in childhood.

Chorea is also closely associated with a rheumatic family history. In the cases in which I was unable to ascertain definite symptoms of rheumatism in the child there was almost invariably evidence of the disease in the parents or in other members of the family. From this it would seem that a common toxic agent exists which is capable of giving rise to choreic manifestations if it affects principally the cerebral cortex, and rheumatic manifestations if the articulations and serous membranes are attacked—an explanation advanced by Hirt and others. Indeed, we may observe both the manifestations of chorea and rheumatism to a marked degree in certain severe cases of rheumatic fever, and the appearance of choreic symptoms in such cases offers a grave prognosis, as they indicate a high degree of toxemia.

A neuropathic family history is found in a large percentage of cases, and epilepsy, insanity or alcoholism in the parents are undoubtedly potent predisposing causes of chorea. In this respect sex also plays an important role, as girls are far more frequently affected than males. Fright is an exciting cause in many cases. No matter to which class the case may belong, this mental trauma acts as a precipitant of the symptoms.

The largest number of cases is seen between the ages of seven to twelve; before the fifth year it is quite rare, and after puberty it usually disappears spontaneously, although cases have been observed in adults. This must not, however, be confounded with Huntingdon's chorea, which is a hereditary disease developing between the thirtieth and fortieth year, and presenting a most unfavorable prognosis.

The *pathology* of chorea is still obscure. As the action of the toxins upon the cerebral hemispheres would in all probability excite only vascular and nutritional changes these are difficult to demonstrate. The frequency of unilateral disturbances early in the course of chorea, the cessation of symptoms during sleep, the blunting of the mental faculties and the occasional psychic disturbances observed, indicate that the gray matter of the cerebral cortex is pre-eminently affected. Organic changes in the structure of the brain may lead to the development of choreiform movements, especially lesions following a cortical hemorrhage. The term "*post-hemiplegic chorea*" has been applied to these cases, but the movements are, strictly speaking, athetoid in character, usually unilateral, not ceasing during sleep, and associated with rigidity and other evidences of organic disease.

Symptoms.—The onset may be sudden or gradual. A severe fright may be followed within the course of a few hours or a day by evidences of extreme restlessness associated with twitching of certain muscles and jerky movements of the extremities. In cases of more gradual onset the child becomes nervous and listless and shows signs of muscular weakness and awkwardness. It drops objects, writes indistinctly, tires readily and often

stumbles and falls down. When the coreiform movements are fully established they will be found to consist of rapid jerkings of single muscles associated with loss of power of coordination in attempting to perform voluntary movements. There is a delay in the carrying out of the voluntary movement and associated movements usually accompany the voluntary effort. Furthermore, the intended act is not successfully carried out and the child is unable to hold the arm or leg in the position attempted. The tongue and facial muscles are similarly affected and there is more or less impairment in the speech. Ludicrous grimaces may be executed, and the child is unable to remain seated quietly in one position for any length of time. The arms are thrown into continuous irregular action and the legs crossed or shifted from one place to another. Voluntary actions are executed with difficulty, being characterized by extreme awkwardness and futility of purpose. Speech may become indistinct and muffled from involvement of the tongue and muscles controlling the larynx (*laryngeal chorea*) that it is difficult to understand the child.

A more gradual development of the symptoms is seen in those cases resulting from overpressure at school, malnutrition following acute illness, or any other neuropathic cause. The child gives indications of gradually-increasing restlessness and awkwardness, the latter condition resulting from the hypotonia and inco-ordination. These phenomena may begin in one extremity or as a unilateral affection, the first symptom being paralytic weakness. The entire body soon becomes involved, and the apparent paralysis may disappear or simply share in the general muscular debility. These cases are described as *paralytic chorea*, monoparesis being the most common type. Church (Church and Peterson, *Nervous and Mental Diseases*) is of the opinion that many of these cases really belong to the neuritides or to a myelitis, or are combinations of these with chorea.

The movements observed in the face are a twitching of the

eyelids and distortion of the mouth. The tongue exhibits marked choreic twitchings in the majority of cases, even in such where movements of the extremities are slight. Sachs (*Nervous Diseases of Children*) places especial diagnostic value on the movements of the tongue and associated facial action in propulsion of this organ, describing these combined movements as the "facies" or chorea. The tongue movements are slow and coarse, and propulsion of the tongue is attended with unnecessarily wide opening of the mouth, raising of the eyelids and eyebrows, and catching of the tongue between the teeth through choreic movement of the masseters.

The head may be turned from side to side and the shoulders alternately raised and lowered, the hands are alternately flexed and extended at the wrist, and the arms are thrown about in an irregular and jerky manner in severe cases. Attempts to control these irregular movements or to perform voluntary acts only intensifies them, and the child may become unable to feed itself or execute other voluntary acts. When the child's hand is taken between the hands of the examiner, the irregular muscular contractions are readily felt. Attempts to control the involuntary movements of the trunk and extremities usually intensifies them. The legs may be so affected by the muscular weakness and incoördination as to render it necessary to put the child to bed. Although sleep may be so disturbed as to exhaust the child to the extreme, and the great restlessness render it necessary to protect the child against falling out of bed, still, in the majority of cases, the movements abate on lying down and disappear entirely during sleep. The latter symptom is pathognomonic of chorea, serving to distinguish it from other motor disturbances.

The *temperature* is normal in most cases; an elevation of several degrees should lead to a suspicion of rheumatism or endocarditis. The *heart* is affected in the majority of cases of chorea. The percentage of endocarditis reported by different writers varies greatly. Heubner found a murmur in 53 per

cent of his cases, but this does not necessarily indicate that all had endocarditis. In hospital cases the percentage is highest, because the severer cases come to the hospital. Nevertheless, one sees cases of severe type in hospital practice in which there is no endocarditis. In my own cases about 23 per cent developed endocarditis while 50 per cent gave a history of rheumatism. Osler examined one hundred and forty cases two years after an attack of chorea and found evidence of organic heart disease in seventy-two of these patients.

In older children mitral disease is closely associated with chorea. Beside organic manifestations, a cardiac neurosis is also encountered, inducing a group of symptoms which disappear with the disease. Both arrhythmia and a systolic murmur may be present, simulating valvular disease; but the murmur varies from day to day in intensity, is not transmitted, the pulmonary second sound is not accentuated, and hypertrophy does not take place. The condition has been called *cardiac chorea*, and is supposed to indicate irregular innervation of the papillary muscles.

The mental state of the child is one of irritability, mental lethargy with deficient memory and power of concentration, and it may even assume a maniacal type of disturbance. Although true mental derangement is rare, it is not unusual to observe a highly exalted psychical state, especially with relapses or acute exacerbations in severe cases. The face becomes flushed; the eyes are brilliant and have a wild, staring expression; there may be alternate crying and laughing or simply crying out, and the general condition becomes greatly aggravated. With proper management such outbreaks are only of short duration, but they may become of serious import when associated with fever and progressive exhaustion, even terminating in coma and death. This constitutes the *choreic status*, which, however, is fortunately seldom encountered.

The *course* of chorea is quite variable. Although usually described as a self-limiting disease, it is, nevertheless, one

which can be controlled to a marked degree by medication, whereby its course may be materially shortened and the symptoms greatly moderated. On the other hand, although complete recovery is the rule, there are numerous instances in which numerous relapses have been noted, or in which the child carries the evidences of chorea to adult life. The average duration can be placed at about from two to three months, always remembering the possibility of relapses, especially in girls. In a series of dispensary cases reported by Bayley (*Trans. Hom. Med. Soc. of Penn.*, 1896) the average duration from the time of onset was 19.4 weeks, and from the time of the beginning treatment it was 12.1 weeks. In private practice the course is usually shorter because treatment can be more satisfactorily carried out.

Diagnosis.—The main source of error in the diagnosis of chorea will arise from confusing it with the motor disturbances of such conditions as *post-hemiplegic chorea* and *athetosis*, which are postplegic movements associated with paralysis of cerebral origin, and those of *Freidreich's ataxia*, *multiple cerebro-spinal sclerosis* and *hysteria*.

The history of the case, the facies of chorea, the characteristic movements and the association of rheumatic symptoms on the one hand and the absence of signs of an organic nervous affection on the other should render the differentiation easy. *Tics* or *habit spasms* are confined to one particular group of muscles, (eyelids, face, head, etc.) and usually result from some form of local or reflex irritation. They have nothing in common with chorea.

Treatment.—As soon as evidences of chorea are observed the child should be taken from school and every effort made to eliminate from its life all excitement and mental and physical strain. The child must be treated with patience and kindness. Parents should be impressed with the fact that it is utterly impossible for the child to control its movements, and that scolding or constantly calling the patient's attention to his condi-

tion will only aggravate the symptoms. Rest in bed is indicated in all grave cases of abrupt onset. All forms of physical exertion must be interdicted and fatigue strictly avoided. A change of scene and climate is often advantageous. In some instances it is best to take the child from its home surroundings and send it to a hospital or put it in the care of a trained nurse. During convalescence I believe judiciously carried out exercises are of great value.

The diet is of importance. Bearing in mind the rheumatic element in these cases, fats, especially cod liver oil and butter, are of decided value. Meats should be cut down, but milk, eggs, cereals and vegetables may be taken liberally.

Extreme restlessness, insomnia and mental excitement call for a warm bath at bedtime. Hot milk is also a valuable adjuvant in these cases.

The remedies from which I have obtained the best results are *belladonna*, *causticum*, *hyoscyamus* and *agaricus*.

Where rheumatic symptoms are prominent, *actea rac.*, *rhustox.* and *sulphur* are frequently indicated and of value. In cases with pronounced psychic disturbances and extreme muscular activity, *hyoscyamus* is useful.

Arsenicum, the chief remedy of the old school, administered in the form of Fowler's solution, and iron are useful when anemia and other conditions pointing to these remedies are prominent symptoms.

Agaricus.—Spasmodic, jerky movements of the extremities and frequent nictitation of the eyelids (*hyos.*). Sensation of coldness and tingling in various parts; paralytic weakness of legs. The active principal of *agaricus* is not *agaricin* but *muscarin*. Bayley speaks of the latter with praise. Personally my experience has been chiefly with *agaricus* in the second and third decimal dilution and it has acted well as a routine remedy in the milder, non-rheumatic cases.

Bell.—Great mental excitement; delirium approaching to a maniacal condition; the face is flushed and the eyes are brilliant

and staring; there is difficulty of speech, and a sensation of dryness and choking in the throat.

Hyoscyamus should be given if *bell.* does not promptly relieve these symptoms, and if there is an incessant throwing about of the arms and a highly frightened behavior of the child.

Caut.—Paralytic chorea with speech defect. The child stands in a limp, relaxed condition; it is hardly able to walk or dress or feed itself; the voice sounds thick and unintelligible, and the tongue is protruded with difficulty. In such cases *causticum* may be relied upon as of distinct clinical value.

Cimicifuga.—Rheumatic pains in the small joints; endocarditis; after suppression of menses.

Coccul.—Right-sided chorea; face puffed and bluish; hands and feet look as if frozen; paralytic symptoms.— (C. G. R.)

Hyos.—Constant twitching of the eyelids; angular gyratory movements, with inco-ordination; misses what he reaches for; silly expression of face, smiling at everything he hears; chorea after debilitating fevers.

Ignatia.—Highly nervous temperament; easily frightened; starts at the slightest noise, irritable temperament. Mild cases, developing after fright.

Nux vom.—Sensation of numbness in the affected parts; frontal headache, constipation, indigestion, irritability and lassitude.

Pulsatilla.—Chlorotic subjects; mild, tearful disposition; functional cardiac disturbances. Chorea developing at the time of puberty.

Sulphur.—Protracted cases with frequent relapses; rheumatic family history; after suppression of eruptions. Other constitutional remedies which may be called for upon purely diathetic indications are *calc. carb.* and *phos.*, *mercurius*, *phosphorus* and *silicea*. The *salicylates* are often useful and will frequently accomplish more for a bad case requiring palliation than *bromides*.

SPASMUS NUTANS; HEAD-NODDING WITH NYSTAGMUS.

The syndrome of rhythmic movements of the head associated with nystagmus is a peculiar condition occasionally encountered in rachitic and otherwise poorly nourished infants. Of late this phenomenon has attracted considerable attention among pediatricists, and a number of cases have been reported in the literature from time to time.

Nystagmus may be the only symptom, or it may be the first symptom, other nervous manifestations, namely, head-nodding and laryngismus stridulus developing later, as occurred in one of my cases. Blepharospasm may also be present (Amberg), and associated movements in the extremities (Ausch) and temporary loss of consciousness (Hadden) have also been observed. As a rule, the movements cease during sleep.

The majority of cases occur in infants under one year. The early signs of rickets are usually present. There is no pathologic lesion, but most probably the symptoms are due to irritation or exhaustion of the nerve centres for the muscles governing these movements. Henoch has pointed out that the nuclei of the oculo-motorius and the nerves governing the movements of the neck are adjacent, and that, therefore, they are readily irritated simultaneously. In many cases there is no doubt as to the exciting cause, namely, keeping the child in a dark room with the eyes exposed to the bright light of a window, analogous to the etiology of miner's nystagmus. All my cases have occurred in dispensary patients from the poor, crowded districts.

The prognosis is favorable, as the symptoms depend partly upon the underlying malnutrition or spasmophilia which may be present in the case. The treatment is purely symptomatic and is to be conducted upon the lines as laid down in the discussion of rickets.

NEUROPATHIC CONSTITUTION AND HYSTERIA.

Many children show distinct evidence of a neuropathic constitution at an early age. The manifestations of such a constitution are an abnormal reaction both in intensity and duration, to physical and emotional stimuli. Certain forms of reflex irritation which a normal child may disregard are likely to produce marked symptoms while the emotional sphere of the child is intensely affected by its environment.

The cause of such an unstable nervous system is mainly hereditary. As a rule the parents are neurotic or there is a family history showing a neuropathic taint. The child's environment is usually responsible for the development of the neuroses and psychoses which are liable to occur in these children. Certain nutritional disorders also disturb the normal balance of the nervous system and spasmophilia, hypertonia, the exudative diathesis and malnutrition with anemia are frequently found as an underlying condition.

Symptoms.—The neuropathic infant is a poor sleeper, cries overmuch and is readily frightened. It is subject to frequent attacks of vomiting and diarrhea. Often outbursts of anger will occur during the first year of life and the infant demands the mother's entire time, day and night. As the infant grows older it becomes headstrong and self-willed. One of the greatest difficulties which these cases present is to make them eat the food selected for them; they will only eat certain things and will rather starve than eat what is set before them. Another difficulty is to make them sleep as much as they should. During sleep they often cry out, jerk and twitch or toss about constantly. They often suffer from night terrors or somnambulism. Many phobias are observed among these children; they are afraid to go to bed in the dark and they may have abnormal fears of certain animals or certain kinds of food. States of mental excitation and depression frequently alternate. They are abnormally imaginative and often given to the fabrication

of long stories of personal adventure (*pseudologia phantastica*). They become truants at school and they are often untruthful although appearing to be unusually bright and often possessing a certain personal charm. Some of the stigmata of hysteria are usually elicited in the careful examination of such children.

HYSTERIA.

Hysteria is a psychoneurosis described by Moebius as "A state in which ideas control the body and produce morbid changes in its functions." Almost every organic disease can be simulated by this peculiar nervous derangement, for which reason its recognition and proper understanding are of the highest clinical importance. Children are by no means exempt from hysteria, and sex bears no etiological relationship to the disorder. Although it may be encountered in early childhood, still it is rare before the tenth year, and most prevalent at the period of puberty and adolescence. Heredity plays an important role, a neuropathic family history being present in most cases. In reviewing the child's life history it will be found that in most cases the manifestations of a neuropathic constitution were already present in early life. As exciting causes, emotional disturbances—especially fright, grief, jealousy, and minor traumatisms in which the mental shock occurring at the time of the accident is entirely out of proportion to the injury sustained—are inseparably linked with hysteria. In the latter instance suggestion also enters into consideration, being one of the strongest influences in causing as well as in removing hysterical phenomena. Reflex irritations, such as tight and adherent foreskin or adherent hood of the clitoris, are mentioned as exciting causes. To these must also be added the baneful influence of improper training and discipline, bad habits and various debilitating illnesses.

Symptoms.—Hysteria in childhood differs from the hysteria of adults in the fact that the manifestations are simpler and more limited in their distribution, thus corresponding to the

immature mental development of the child. We rarely find the puzzling symptom complexes observed in the adult. Hysteria in childhood is usually monosymptomatic and when paralysis occurs it is more likely of a single extremity than hemiplegic or paraplegic as in adults.

Stigmata.—The *mental condition* is characterized by diminished will power, loss of memory and lack of determination, and indecision. Impressionability and irritability characterize the temperament. These subjects are very susceptible to suggestions, and the mood vacillates between sadness and gayety, uncontrollable paroxysms of alternate laughing and crying being a frequent occurrence.

Disturbances of sensibility are encountered as complete or partial cutaneous anesthesia, or hyperesthesia in certain localities. It is usually found in parts which are paralyzed, a hemiplegia with anesthesia being strongly indicative of hysteria. Irregular islets of anesthesia are likewise characteristic of hysteria. The area of anesthesia does not correspond with the distribution of special nerve trunks or to the areas of sensation supplied by the different spinal segments, but seems to conform rather to the cortical representation of sensory areas. The mucous membranes may be anesthetic and the special senses become perverted or abolished, leading to disturbances of sight, hearing, etc., or sudden blindness or deafness. The throat may become anesthetic, so that we can irritate the fauces without producing gagging. Likewise, anesthesia of the nose, conjunctiva, larynx, etc., is to be encountered. The reflexes are not disturbed, as they are in organic lesions associated with anesthesia.

The *motor disturbances* to be observed are a general retardation of voluntary movements and muscular weakness and incoordination. This is explained by the presence of anesthesia and loss of muscular sense and of the power of mental concentration.

To the milder forms of spasmodic affections belong, notably,

globus hystericus; hysterical cough, hiccough and glottic spasm; spasm confined to certain muscle groups, notably those of the neck.

Accidents.—To the *accidents* of hysteria belong certain transitory disturbances manifesting themselves as convulsive seizures (*grand attacks*; *hystero-epilepsy*), or as motor and sensory disturbances of major degree, closely simulating a variety of organic diseases.

Grand attacks belong to the rarer forms of hysteria in childhood; but as they bear a superficial resemblance to epilepsy, they will be considered in full. The attack is preceded by depression of spirits and a sensory aura, most commonly the *globus hystericus*. This is described by the patient as the sensation of a ball rising into the throat and is due to spasmodic contraction of the pharynx and esophagus. A general tonic spasm, which persists for a few minutes, marks the first stage of the attack. The child lies stretched out, with the limbs extended and rigid, the fingers and toes being flexed. Slow, rigid movements of wider range executed by the arms, and flexion and extension of the feet, may be observed during this stage. The jaws are tightly closed, and respiration is slow and irregular or entirely suspended. The face assumes a bloated appearance, and the veins of the neck are prominent and swollen.

The clonic stage is ushered in by short, jerky movements involving the face and extremities. These movements increase in severity, but do not assume the regular clonic type of epilepsy, being more irregular and of a struggling character. Respiration becomes interrupted and sobbing. Biting of the tongue is rare, as is also involuntary defecation and micturition. After the course of a few minutes the movements cease abruptly, and a period of resolution or repose sets in—a condition simulating sleep. This may end the attack, or be succeeded by cataleptic manifestations, during which the patient becomes fixed in a variety of rigid postures. Extreme opisthotonos is

a common position observed in hysteria. A phase of large movements now follows, in which the subject may cry out in fear or rage and strike or bite at those about him. Peculiar sounds are sometimes uttered, resembling, for instance, the barking of a dog, and, when associated with the above symptoms, constitute spurious hydrophobia.

The period of passional attitudes observed in adults is very rarely seen in children. The period of delirium, in which the child sobs and pleads in a pitiful manner, or expresses various hallucinations, often terminates the attack, after consciousness is restored.

Motor accidents occur as paralysis and contractures. They are usually of sudden onset, as the result of fright or injury; less commonly they develop gradually. Hysterical paralyses correspond in their general characteristics with those of central origin but there is a greater tendency to contractures and anesthesia and in cases of the leg there is a negative Babinski's sign. Monoplegias are more common than hemiplegia or paraplegia. Sometimes it is confined to a joint and simulates chronic arthritis. The paralyzed part is frequently anesthetic, and the anesthesia corresponds to the cortical distribution of sensation, not being confined to one or more nerve trunks, as in peripheral nerve and spinal affections.

In hemiplegia the face escapes, with the exception of the eye-muscles, which are at times affected. Anesthesia is common, while in organic cerebral hemiplegia it is rare. Again, the contractures of hysteria partake more of the nature of spasmodic voluntary resistance, and atrophy never takes place excepting as a slight amount of wasting resulting from non-use. Loss of power is not absolute, and the degree of paralysis may vary from day to day. The gait also differs from that observed in cerebral palsies in that the leg is dragged along in a limp condition, not being swung out in a lateral direction, by which the foot is made to describe an arc.

A characteristic form of hysterical paralysis observed in

children is *astasia abasia*. In this condition the child can move its legs while lying down and reflexes are normal but he is unable to walk or stand.

Contractures may exist independently or in association with paralysis and anesthesia. The extremities are most frequently affected. When the hands and feet are affected, the fingers and toes are flexed. With involvement of the larger joints there is extension, so that the arm and leg are held out straight. Contractures may occur in monoplegic, hemiplegic or paraplegic distribution. In deep sleep the rigidity usually disappears.

Hysterical coxalgia is a most important subject presenting itself for consideration to the pediatricist. No doubt, the numerous cases of so-called reflex paralysis and coxalgia reported as having been cured by circumcision belong to this category. Apparently, every subjective and objective symptom of hip-joint disease has been mimicked by this neurosis, and nothing short of a careful examination under an anesthetic will serve to differentiate such a case from true hip-joint disease. This holds good for other joint affections in which fixation and pain without any objective signs are present. An X-ray examination will show the bones and joint surfaces in a normal condition. Under the influence of an anesthetic the rigidity of the joint disappears.

Sensory Accidents.—A *pseudo-meningitis* is occasionally encountered, and is distinguished from true meningitis by the history of the case, the absence of slowing or irregularity of the pulse and active pupils. In other respects it bears a close similarity to meningitis, presenting intense headache; vomiting; fever; vasomotor streaks (*taches cérébrales*), and rigidity of the neck and extremities. A lumbar puncture will serve to exclude meningitis.

Spinal tenderness may be confined to the region of a few vertebræ and closely simulate Pott's disease; but if the patient's attention can be detracted momentarily a considerable amount of pressure will be borne without causing pain.

Visceral Accidents.—Disturbances in the respiratory tract show themselves as *aphonia*, usually developing suddenly after a fright, the voice being lost, but cough persisting; *dyspnea*, due to laryngeal or diaphragmatic spasm; *tachypnea*, sudden attacks of extremely rapid breathing, presenting alarming symptoms, without the evidence of physical signs to account for the same.

In the digestive tract, *vomiting*, *globus hystericus*, *esophageal spasm*, *anorexia* and *obstinate* constipation are to be observed.

Frequent urination of large quantities of pale, limpid urine or complete anuria, sometimes retention of urine, are the disturbances encountered in the urinary tract.

The *prognosis* of hysteria is not unfavorable in children as they are readily influenced by suggestion, and, if the proper surroundings and intelligent treatment can be provided, recovery is generally comparatively rapid. The accidental disturbances, as a rule, disappear spontaneously after a variable period of months or year, or they may come and go. The mental state can, however, seldom be improved beyond a certain limit, and the hysterical temperament will persist throughout life in the majority of cases.

Sensory accidents are stubborn in their course, bringing considerable suffering to the patient and much anxiety to the friends and attendants. The spasmodic manifestations can usually be cured promptly if the patients can be taken from their parents and kept under intelligent supervision.

In the *diagnosis* much importance is to be attached to a recognition of the stigmata of hysteria; in other words, the hysterical temperament, in conjunction with the emotional origin of the ailment and the polymorphous and changeable character of the manifestations. Besides, the differential features serving to separate hysterical from organic diseases, as pointed out in the symptomatology, should serve in the differential diagnosis from organic affections.

Treatment.—The general management of hysteria resolves itself into removing all exciting causes, isolation being the most effectual method for this purpose; attending to the removing of all sources of reflex irritations, such as phimosis, adenoids and errors of refraction, and building up the constitution by means of regular calisthenic exercises, a highly nutritious diet and a liberal amount of sleep.

Suggestion is a most potent agent in restoring the patient's confidence and overcoming the various disturbances which have an imaginary origin. In managing cases of paralysis our main effort must be in the direction of promising the patient that the line of treatment employed will bring positive results. To emphasize this suggestion such adjuvants as massage and electricity are employed with benefit. This does not, however, apply to ill-managed cases of long standing, in which the surgeon's aid may have to be sought.

The beneficial results following upon even the most trivial surgical measures resorted to in hysterical subjects is a noteworthy clinical fact, which often can be taken advantage of as a justifiable means of treatment.

Medicinal treatment serves a two-fold purpose, namely, by augmenting the force of the suggestions and also by improving the patient's general condition. It is needless to mention the close relationship existing between neurasthenia and hysteria in children, and, therefore, remedies which will improve the nutrition of the nervous system cannot fail to influence the hysteria. Such remedies as *picric acid*, *calcareo carb.*, *silicea* and *phosphorus* exert a potent influence in this direction.

Remedies possessing notably hysterical symptoms are *ignatia*, *hyoscyamus*, *aconite*, *asafoetida*, *moschus* and *valerian*. The efficiency of drugs in such conditions as hysterical palsy and hystero-epilepsy is doubted by many. Arndt (*Practice of Medicine*) expresses the opinion that "they are often helpful, especially in times of great emotional excitement."

An unfortunate error often made in the care of hysterical

subjects is to look upon them as simply imagining their troubles and, therefore, requiring no treatment. Nowhere more than in hysteria does it require firm yet kindly supervision and persistent and encouraging suggestion to lift the patient out of his imaginary fears and afflictions. With a hysterical child we have a campaign of education before us which must be continued to adult life.

PARALYTIC AFFECTIONS; CEREBRAL PALSIES.

The *cerebral palsies* of childhood comprise a group of conditions which may be either of intra-uterine onset, or which are acquired during parturition or at a still later period. Cases of intra-uterine origin are usually developmental in character, and to this group belong porencephalia, agenesis corticalis and other defects, although evidences of hemorrhage and sclerotic changes, as a result of traumatism, fetal meningo-encephalitis and syphilis, have been observed in rare instances.

In birth-palsies, hemorrhage is the primary lesion. It occurs frequently in protracted labors, and although forceps-pressure may directly induce a hemorrhage, still it does not play as important a role as long-continued compression of the head in the pelvic straits or within the uterus. It has also been supposed that undue pressure upon the trunk during the extraction of a breech presentation may be the direct cause for the rupture of a bloodvessel in the brain. Cerebral hemorrhage is especially likely to occur in a precipitate labor of a large infant. The bleeding takes place from the capillaries and veinules of the pia mater or choroid plexus in most cases, more rarely from the longitudinal sinus and veins, and almost never from an artery. Venous congestion attending compression of the cord and asphyxia may give rise to a pial hemorrhage, but the weight of evidence is in favor of attributing the majority of cases of asphyxia neonatorum to hemorrhage. A new-born infant therefore, with pallid asphyxia should be looked upon as most likely an apoplectic one unless good reasons for some other cause are at hand.

Where the amount of blood-extravasation is not sufficient to cause death, it ultimately is absorbed or becomes organized with consequent sclerosis of adjacent areas of brain-substance and developmental retardation. The symptoms attending such a condition will naturally depend on the locality affected.

The cerebral palsies encountered later in child-life are the result of either hemorrhage, embolism or thrombosis. A cerebral abscess or tumor may likewise cause definite paralytic manifestations, but in their etiology and clinical course they differ distinctly from the foregoing conditions. Hemorrhage at this period of life is more frequently meningeal than cerebral. It may result from traumatism, arteritis, or from a sudden and severe venous congestion of the brain occurring during a convulsion or during a paroxysm of whooping-cough.

Birth-palsies are usually bilateral, that is, diplegic or paraplegic, while the later palsies are most frequently hemiplegic. Sometimes hemiplegia attacks an infant in apparently perfect health, the symptoms coming on with fever, followed by convulsions and hemiparalysis. Strumpell first advanced the theory that these cases were inflammatory in character, due to an acute infection. The majority are examples of *polioencephalitis*, or the cerebral type of infantile paralysis.

Abscess is most frequently secondary to suppurating otitis media.

Sinus thrombosis results from extreme anemia in conjunction with feeble heart's action occurring during exhausting illness, or from infection from the middle ear. In such cases thrombosis of one of the lateral sinuses, with its characteristic symptoms, results. *Embolism* is most frequently associated with endocarditis, only in rare instances originating from clots which have formed in the left auricle or elsewhere.

Symptoms.—The lesions just enumerated may be productive of a variety of manifestations, for which reason we may encounter either *hemiplegia*, *diplegia*, *paraplegia* or *monoplegia* in these cases. The last two are rare, especially monoplegia,

and paraplegia is frequently only apparent—a careful examination also revealing evidences of paralysis in the arms together with mental deficiency.

The mental condition is impaired and the head is usually small or irregular in form. Epilepsy develops in many of these cases, assuming the true degenerate type of the disease.

Diplegic cases are congenital, or result from injuries sustained during parturition. As above stated, the lower extremities are most markedly affected, and athetosis is a prominent symptom. Lack of mental development can be traced back to the earliest period of infancy and on account of the spasticity of the legs they do not learn to walk until very late. The rigidity in both arms and legs varies in degree; when pronounced it reminds one of the resistance encountered in bending a piece of lead, for which reason it has been described as “lead-pipe rigidity.” Together with this there is a crossing of the lower extremities due to adductor spasm and a tendency to equino-varus. The gait is, therefore, extremely difficult or impossible, and the hands are usually not well under control, being entirely helpless when athetosis is marked. A type of congenital diplegia resulting from defective development of the pyramidal tracts in the brain and cord, seen in under-developed or premature children, has been described by Little, of London (*Little's Disease*). Children afflicted with Little's disease are not deficient in mind, and the spastic condition usually improves with the development of the nervous system.

Sachs (*New York Medical Jour.*, May, 1896) has reported a series of cases of congenital cerebral agenesis occurring as a family disease, in which amaurosis, progressive debility and a fatal termination are the clinical features. More or less diplegia, with spasticity, is usually present. A number of these cases is reported in the literature under the name of “*Amaurotic Family Idiocy*.”

The *prognosis* is unfavorable in all cases, but especially in the diplegic forms, in which little can be done aside from im-

proving the child's general state by means of massage and faradism, or by surgical measures when necessary. The proper training of such cases is, however, of the greatest importance, through which means both the mind and body may often be wonderfully improved.

ACUTE POLIOMYELITIS, OR INFANTILE PARALYSIS.

The term "Infantile Paralysis" expresses the popular conception of the specific acute infectious disease first systematically studied and accurately described by Jacob von Heine in 1840. It was long believed that this disease was a primary affection of the gray matter of the cord, whence the term "poliomyelitis." The study of old cases in which atrophy of the anterior horns of the spinal cord was found, was responsible for this view. Our present conception of poliomyelitis begins with the masterful study of the great epidemic in Sweden in 1905 by Wickman. The pathological and clinical studies of Wickman showed conclusively that poliomyelitis is an acute infectious disease which is evidently spread by contact of healthy children with infected ones, and that one of the chief dangers of the rapid spread of an epidemic is the large number of abortive, or non-paralytic cases prevalent during an epidemic. His studies also demonstrated the fact that the disease is accompanied by a pathological reaction throughout the entire body, and that the lesions in the cord are but part of a general inflammatory reaction which sometimes involves the brain as well as the cord.

Etiology.—Poliomyelitis occurs both endemically and epidemically. In large communities we may expect to encounter sporadic cases at any season of the year. The striking characteristic of the disease, however, is its occurrence in epidemics; these usually reach their highest point of development during the hot summer months. Winter epidemics have occurred in Norway and Sweden. Whether insects are responsible for the spread of the virus has not been definitely determined. The

virus has been demonstrated in the secretions from the nose and throat of infected individuals and also in the stools. Sheppard (*N. Y. State Jour. of Medicine*, 1916, XVI, 442) has reported several instances of group infection which seem to prove conclusively that the disease may be transferred by human contact.

Age is an important etiological factor. The great majority of cases occur in children from two to five years old. In some epidemics many adults have been attacked. Draper (*Acute Poliomyelitis*, Blakiston's Son & Co., 1917) is of the impression that a certain type of child is especially susceptible to this infection. He calls attention to the large number of well-grown, plump, broad browed and broad and round faced children one sees attacked during an epidemic.

The Organism.—Landsteiner and Popper in 1909 succeeded in transferring the disease to monkeys but were unable to propagate the infection. Shortly after their experiments were reported Flexner and Lewis succeeded in propagating the infection indefinitely from monkey to monkey by means of intracerebral inoculations. In 1913 Flexner and Noguchi reported the successful cultivation of a filtrable virus with which they were able to reproduce the disease in monkeys. Recently a pleomorphic coccus has been isolated from the brain and cord after death from poliomyelitis by Mathers (*Jour. Amer. Med. Ass.*, Sept. 30, 1916). This organism caused paralysis in rabbits and monkeys. Rosenow and others working independently report finding a similar streptococcus-like organism from the throat and tonsils as well as from the nervous system. Whether this organism is a secondary invader or the actual cause of poliomyelitis and capable of existing in filterable and nonfilterable forms has not been finally settled.

Pathology.—Recent studies of fatal cases, and a close scrutiny of the symptoms of poliomyelitis, especially during the epidemics in which many atypical forms of the disease are encountered, show that poliomyelitis presents the character-

istics of a general infection, and that the meninges of the cord and sometimes of the brain are distinctly involved. It is now assumed that the infection gains access from the upper respiratory tract, possibly through the nasal cavity, and is carried to the cerebro-spinal nervous system by means of the lymphatics. The earliest changes observed are hyperemia and edema of the meninges with exudation into the arachnoid spaces (acute interstitial meningitis). This is most marked on the anterior surface of the cord and when the process extends into the cord, it does so along the blood vessels which enter the anterior fissure and supply the anterior horn of the cord. There is an associated infiltration of the meninges and medulla with mononuclear cells. This is most marked about the blood-vessels and may cut off the circulation from the nerve cells with resulting necrosis of the affected area. Sometimes hemorrhagic changes occur.

Areas that have been affected by pressure only may regain their function after the hyperemia subsides and the cellular exudate is absorbed. Such areas, however, which have suffered from necrosis or extensive hemorrhage will undergo atrophy and present the typical appearance of the shrunken anterior horns mentioned in the older descriptions of the disease.

Symptoms.—Poliomyelitis may present a diversity of clinical manifestations, since it not only varies widely in its severity, ranging from abortive, non-paralytic cases to fulminating, rapidly fatal cases, but also depending upon which region of the nervous system is mainly attacked. The following clinical types of poliomyelitis are recognized:

(1) *The Abortive Type.*—Many cases of this type occur during epidemics and render control difficult. These cases are true instances of poliomyelitis, but lack the characteristic paralysis of the distinctly spinal type. Definite indications of meningeal involvement are, however, usually present.

The attack may be ushered in by gastrointestinal symptoms, vomiting and diarrhea, or with coryza, cough, general malaise,

pains in the extremities. Moderate fever persisting for several days is present. Rigidity of the neck and Kernig's sign may be present, verifying the presence of meningeal involvement. Rigidity of the spine can also be elicited in many of these cases as well as tenderness of the muscles of the extremities and transient palsies. A lumbar puncture will verify the diagnosis when such suspicious symptoms are present.

(2) *The Spinal Type*.—This represents the typical cases of poliomyelitis. The period of incubation is stated to be from five to ten days. A prodromal period is observed in most cases, the symptoms being referred to either the upper respiratory tract, the tonsils or the gastrointestinal tract. A characteristic feature of poliomyelitis is the fact that there is frequently an interval of several days of remission of symptoms between the occurrence of the prodromata and the initial symptoms of involvement of the spinal cord. Draper likens this arrangement of the two masses or humps of symptoms to the dromedary's back and speaks of the cases presenting this peculiarity as the "dromedary type." He is of the opinion that during an epidemic many children who are infected with poliomyelitis virus develop only the first group of symptoms because the virus gains entrance with difficulty to the central nervous system. The remission in paralytic cases indicates the time required for the virus to reach the cord. Cases of sudden onset may be regarded as examples of the malady in which the prodromal, or systemic stage has been overlooked or forgotten on account of its triviality.

The onset is characterized by fever, rapid pulse, rapid respirations and restlessness. Vomiting is a common occurrence at this stage; convulsions are less common. Rapid pulse and sweating are very suggestive of poliomyelitis especially if there is associated the spinal rigidity so characteristic of the early stage of the disease. There may be an associated coryza, pharyngitis or diarrhea.

The temperature is usually not high, averaging from 101°

to 103°. The pulse, however, is disproportionately rapid. Cases with involvement of the cervical portion of the cord have, in my experience, shown a slow pulse.

The earliest symptoms referable to the nervous system are drowsiness and apathy associated with a characteristic nervous irritability. The child resents being touched or examined and wants to be left alone. As soon as the paralysis develops it becomes very fretful, complaining and cannot be made comfortable.

Paralysis usually develops early; in the majority of cases the lower extremities will show signs of involvement on the first or second day after the fever has set in. It may not reach its maximum until the third or fourth day. Paralysis is rarely delayed beyond the sixth day. One of the gravest dangers of poliomyelitis lies in the possibility of the paralysis ascending to the centres controlling the respiratory muscles, with resulting death from asphyxia. This sometimes occurs several days after the onset of the first signs of the leg paralysis.

In the majority of instances the paralysis remains limited to the legs; sometimes the abdominal muscles and the muscles of the back are also involved. As the disease subsides it is generally found that one side is much more affected than the other. Involvement of the arms is rare. The astonishing feature of the disease is the rapidity with which the paralysis clears up during convalescence. Cases which presented a complete paraplegia during the height of the disease may be left with very little permanent paralysis. This is readily explained by the pathology of the affliction. From a study of the distribution of the lesions in 868 cases reported by Wickman it was found that paralysis was limited to one or both legs in 353 instances and to a combination of arms and legs in 152 instances. The arms were affected alone in only 75 cases while the trunk muscles were involved in conjunction with either the arms or legs in about 100 cases.

The paralytic condition remains stationary for a period of

from two to three weeks. At the end of this time spontaneous improvement sets in and a gradual improvement in the paralyzed muscles continues for several months. After that time improvement may continue but at a much slower rate and then only under appropriate treatment. Some improvement may still be anticipated as late as two years after the attack.

The affected limbs present the flaccid type of paralysis resulting from involvement of the lower motor neurons with loss of the tendon reflexes. Muscles that are completely and permanently paralyzed undergo marked atrophy; sometimes the growth of the entire limb is retarded. As a rule, however, muscles are only partially paralyzed and the fibres which have escaped become hypertrophied and compensate for the defect. The extensors are more often involved than the flexors. There is early loss of faradic irritability in the completely paralyzed muscles and the reaction of degeneration can be elicited in them.

(3) *Bulbospinal Type*.—This is a variety of the spinal type in which the cranial nerve centers in the medulla become involved. Usually it presents the terminal stage of the rapidly fatal ascending cases, simulating Landry's paralysis, in which the diaphragm and intercostal muscles are also involved. Sometimes bulbar symptoms occur early, notably difficulty of deglutition and hoarseness. These cases are very serious but occasionally recovery takes place. Strabismus and facial paralysis are other frequent manifestations of this type and these cases also may recover; residual paralysis, however, usually remains. Bulbar symptoms may exist without any spinal involvement, although this is rare.

(4) *Cerebral Type*.—This type was first described by Strumpell, who recognized the clinical identity of polioencephalitis and poliomyelitis. In these cases the virus of the disease spends its effect upon the cortical motor areas of the brain instead of upon the gray matter of the cord and an upper motor neuron paralysis results. The resulting symptoms are a hemiplegia with spasticity, heightened reflexes and no atrophy.

The attack is usually ushered in with fever, vomiting, convulsions and delirium. Sometimes the symptoms closely simulate meningitis, which can only be differentiated by the lumbar puncture findings and the discovery of the associated hemiplegia. The prognosis is more grave than in the purely spinal type but it is not as fatal as other forms of meningitis.

Diagnosis.—The diagnosis of poliomyelitis is readily made in the frank spinal type, the rapidly developing extensive paralysis of the legs with or without involvement of the arms and trunk muscles; the paralysis being of the flaccid type with lost reflexes and promptly followed by muscular atrophy, stamps the case distinctly as one of infantile spinal paralysis. The atypical forms may present many diagnostic difficulties, however, especially the abortive types. This is particularly true in sporadic cases. The cerebral and meningeal types are frequently confused with meningitis and the differentiation can at times only be made by means of a lumbar puncture. Lethargic encephalitis in its early stages may also be confused with the meningeal type, especially since the lumbar puncture findings are similar in these two conditions.

The characteristics of the cerebrospinal fluid in poliomyelitis are a clear fluid containing a high percentage of polynuclear cells in the first two or three days (preparalytic stage) of the disease. These are rapidly replaced by mononuclear cells. The average cell count is from 75 to 200. Another characteristic of the fluid is its power to reduce Fehling's solution; this is fairly constant and is not found in tuberculous meningitis. Globulin is markedly increased. Sometimes the fluid presents a characteristic yellowish discoloration.

Treatment.—Absolute rest in bed during the acute inflammatory stage is imperative. It is not wise to resort to massage or electrical treatment until pain and tenderness have disappeared from the affected limbs and until the temperature has been normal for at least a week. The paralyzed limbs should at once be maintained in a proper position by means of pillows

and sand bags when necessary in order to prevent overstretching of the paralyzed muscles and heat should be applied to keep the limbs warm.

At the expiration of about three or four weeks active treatment with electricity and judicious massage may be begun. If the muscles do not respond to the faradic current, the galvanic should be employed. The object is to produce muscular contractions in order to improve the nutrition of the muscle and restore function as far as that is possible. Passive movements should be added to the treatment in order to overcome deformities. When once established, these will require surgical measures to correct them. The disability in a joint resulting from atrophy of one of the muscles either flexing or extending the same, is often satisfactorily corrected by a properly adjusted brace, which not only supports the joint but also prevents deformity.

The remedies indicated in the early stages are *acon.*, *bell.*, *bry.*, *gels.* and *rhus tox.* These are indicated by their influence over inflammatory processes in general and on account of their specific action upon the cerebrospinal nervous system and its investments.

Belladonna corresponds both symptomatically and pathologically to the stage of onset of the disease. The fever with hot skin, disproportionately rapid pulse, flushed face and bright glassy eyes, are strong indications for this remedy.

When paralysis develops, *gelsemium* should be employed. This remedy has been extensively used in the homeopathic school with success and has been recommended recently by some old school writers.

During convalescence *mercurius* may be given for the purpose of stimulating absorption of the exudate. If the affected limbs are painful and tender *bryonia* is preferable.

For the residual paralysis, *causticum* is of value. In numerous instances I have seen improved tone in the paralyzed muscles follow its administration.

Serum therapy—The best results obtained from serum therapy have been with the use of human immune serum used both intra-spinally and intra-venously. Human serum, however, is difficult to obtain and we therefore welcome a horse serum which promises to give definite curative results.

Such a serum has recently been prepared by Rosenow by repeated injections of the pleomorphic streptococcus isolated by him from cases of poliomyelitis but it is still too early to judge the results from the use of the same.

PROGRESSIVE MUSCULAR ATROPHY.

The Idiopathic Muscular Dystrophies bear a close outward resemblance to the late manifestations of anterior poliomyelitis. They have been divided into a variety of clinical types, but are all closely related both etiologically and pathologically. The main point of distinction between these myopathies and poliomyelities is their slow and progressive development, the symmetrical distribution of the atrophic changes, and the hereditary factor in their etiology.

The *pathological changes* observed in progressive muscular atrophy take place primarily in the muscles themselves, any changes found in the cord being looked upon as secondary. The muscle-fibres at first become hypertrophied, undergoing subsequent atrophy. The connective tissue is slightly increased.

The following types are recognized:

The Juvenile Type of Erb.—In this form the muscles of the arms and shoulders are mainly affected.

The Facio-scapulo-humeral Type of Landouzy-Déjérine (Infantile Form of Duchenne), in which the face, together with the arms and shoulders, is affected.

The Peroneal Type of Charcot and Marie, in which the peroneal muscles become atrophied. This may be followed by atrophic changes invading the legs, trunk and upper extremities, and there is evidence of cord-lesions associated with the atrophy,

showing itself as fibrillary twitching and reaction of degeneration.

Pseudo-hypertrophic Paralysis is a disease of early childhood most frequently seen in boys, characterized by enlargement of the calves and buttocks, associated with atrophic changes. The muscles finally shrink, presenting the same condition as the other forms of atrophy. The characteristic symptoms produced are a waddling gait; difficulty of climbing up stairs and great awkwardness; enlargement of the legs and buttocks; lordosis; inability to rise from the ground without the aid of the hands. In order to attain the erect position the child supports the hands on the anterior surface of the thighs and gradually pushes himself upright.

FAMILY ATAXIA.

Family ataxia, also known as *Friedreich's disease*, occurs as a family disease, several or all of the children being attacked by a degenerative process of the posterior and lateral columns of the spinal cord. The cord lesion is a neuroglial sclerosis evidently an indication of a developmental defect. The first symptoms usually make their appearance shortly before puberty, a period at which the process of growth and nutrition are taxed to their utmost. When there are successive cases in a family they usually develop at a progressively increasing earlier period of life. An acute infectious disease may also hasten the development of symptoms, and for this reason the first symptoms may occur in early childhood following an acute illness.

Hereditary cerebellar ataxia of Marie is characterized by a similar defective condition involving the cerebellum; but it develops after puberty, and is accompanied by pronounced choreiform movements, increased deep reflexes, and optic nerve atrophy, symptoms not found in spinal ataxia.

Symptoms.—One of the earliest symptoms noticed is an awkwardness in the legs, marking the beginning of the ataxia. Later the arms become involved. There is first unsteadiness in walking and standing, the child sways from side to side in

attempting to maintain its equilibrium. As the muscular sense is not lost, the condition depending entirely upon inco-ordination, no increased difficulty in standing is noticed when the eyes are closed (absent Romberg sign). The ataxia is associated with gradually increasing loss of power. The knee-jerk is lost early in the disease (Westphal's sign). This distinguishes it from the cerebellar variety, in which there is also at times an ankle clonus.

Disturbances of speech develop as inco-ordination becomes general. The speech is irregular and jerky, and lacks modulation and rhythm.

Nystagmus may develop later in the disease, being especially noticed with lateral rotation of the eyes. The expression is one of apathy and indifference, although the intelligence is not impaired early, but it is retarded with the progress of the case, as is also the physical development. Shortening of the foot, with exaggerated plantar arch and retraction of the great toe (*club-foot* and *hammer-toe*), is a common deformity of family ataxia. Another deformity is dorso-lumbar scoliosis. These deformities may develop before ataxia becomes pronounced, and constitute early evidence of the disease.

The *course* is that of a progressively-increasing and hopeless malady, but remissions or aggravations may take place. There is nothing in the disease itself to cause death, for which reason the person so afflicted may live to adult life.

Isolated cases must be differentiated from *cerebellar ataxia*, *chorea* and *multiple (insular) sclerosis*. In the latter there is characteristically scanning speech, spastic gait and intention tremor.

SYRINGOMYELIA.

Syringomyelia is a disease of the spinal cord in which the spinal canal becomes pathologically enlarged as a result of the break down of a gliomatous infiltration. By the same process new canals of considerable length may be found within the gray matter of the cord. It is a rare affection in early childhood.

The etiology is obscure and nothing definite is known, excepting that embryonal neuroglial tissue degenerates or becomes the seat of hemorrhage.

The symptoms resulting from a central myelitis or from a hemorrhage into the cord—the latter, at times, occurring during parturition—cannot be distinguished from those belonging to glioma.

Symptoms.—The disturbances of syringomelia may be divided into several groups. Involvement of the sensory pathway in the gray commissure and posterior horns and columns gives rise to loss of pain and heat perception, without, however, loss of the tactile sense. This anesthesia may be so complete and extensive as to render the patient insensible to almost any kind of pain and expose him to many dangers.

Motor disturbances develop later than the sensory, and present paralysis of groups of muscles of a limb, usually becoming bilateral and accompanied by trophic changes. The reaction of degeneration is present. These symptoms indicate involvement of the anterior horns and pyramidal tracts.

Vasomotor disturbances, cyanosis, coldness, cutaneous eruptions and dermatographia may accompany the above process. Trophic changes, with resulting atrophy, fragility of bones, enlargement of the hands, and tendency to the development of whitlow and abscesses, are also to be noted.

The *course* is progressive, and results fatally when bulbar crises set in. In the *diagnosis*, the *idiopathic muscular dystrophies*, *hysteria* and *multiple neuritis*, are to be differentiated. The distinct features of syringomyelia are its gradual development and insidious onset, and the dissociation of touch and pain in conjunction with motor, trophic and vasomotor disturbances.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

Multiple of *disseminated* sclerosis, as the name implies, is a degenerative process affecting the brain and cord in an

irregularly scattered sclerotic progress. The islets of sclerosis are found principally in the centrum ovale, crus, pons and medulla in the brain, and in the cord they are irregularly scattered, as a rule attacking the white matter more prominently than the gray. It is most common between the ages of twenty and thirty, but it may occur in children or even be congenital.

The *cause* of multiple sclerosis is probably to be found in an infection, but, judging from the numerous and often mixed infections noted, it seems unlikely that we have to deal with a specific organism.—(Church).

Symptoms.—Owing to the widely-distributed lesions of multiple sclerosis a variety of clinical manifestations are observed in this disease. The characteristic and most prominent features are:

(a) *Motor.*—A coarse, jerky inco-ordination, especially in the arms, observed on attempts at voluntary movements. This intention tremor is associated with progressively increasing loss of power. The gait is spastic and is associated with de-ranged equilibrium.

(b) *Sensory* disturbances are practically confined to the eye. Nystagmus is a frequent symptom, and optic neuritis and atrophy may develop.

(c) *Cerebral disturbances.*—The speech defect, known as “scanning speech,” in which there is an undue separation and accentuation of the syllables of words, and a state of indifference, loss of memory and dejection, are the prominent cerebral features of the disease. A predisposition to hysteria seems to exist, and it is not uncommon to find hysterical manifestations complicating multiple sclerosis.

(d) The deep *reflexes* are exaggerated, as a rule, but there may be a loss of knee-jerk, and paralysis of cranial nerves in some cases.

The *clinical course* of multiple sclerosis is quite irregular. It may begin gradually and increase in a progressive manner, or it may begin abruptly as an apoplectiform attack, or with

vertigo or visual disturbances. Remissions are not infrequent, and may lead to a belief that the disease has been checked; but complete recovery must be very rare, although Church considers it possible.

Diagnosis.—Multiple sclerosis is to be differentiated from *infantile cerebral palsy*, *hysteria* and *family ataxia*. In *infantile cerebral palsy* the history of traumatism during birth and the early appearance of diplegia, followed by mental retardation, rigidity and athetosis, will serve as a distinguishing feature. In *hysteria* the mental stigmata, the absence of nystagmus, and the presence of sensory disturbances and muscular rigidity, are of great significance, although both diseases may be associated in the same patient. In *family ataxia* there is inco-ordination and spasmodic muscular action; the knee-jerks are abolished, the muscles are flaccid, and the eyes are seldom affected, except by a slight degree of nystagmus, with lateral rotation of the eyes.

The *treatment* of these cases is very unsatisfactory. According to Arndt, *arsenicum* is of especial value. *Tarantula* has also been recommended. Bartlett refers to the salts of *gold*, *lead* and *mercury*.

MYATONIA CONGENITA.

Myatonia congenita, or *amyotonia*, is a congenital affection in which the muscles of the extremities, especially those of the legs are abnormally flaccid and incapable of voluntary contraction. The disease was first described by Oppenheim in 1900 and is sometimes called Oppenheim's disease. The infant is limp and helpless and the legs are useless as in the case of a spinal paralysis. The knee-jerks are either absent or much diminished. The electrical reactions are diminished but there is no reaction of degeneration. The diaphragm escapes but the chest muscles are involved and the thorax presents a characteristic deformity simulating that found in rickets. As a rule some retardation in the child's mental development is also noted.

The *pathological process* is confined to the affected muscles. These show a marked diminution in the size of the muscle fibres. The findings in the peripheral nerves and in the cord are negative.

The *diagnosis* rests upon the congenital character of the affection and the absence of signs of a spinal lesion. The deformity of the chest is characteristic; rickets can be ruled out because this disease rarely develops before the end of the first year. Myatonia should not be confused with *myotonia*, or Thomsen's disease which is an hereditary affection characterized by stiffness and rigidity of the voluntary muscles occurring during voluntary efforts.

MULTIPLE NEURITIS.

Inflammation of several nerves occurring coincidently or in quick succession occurs mainly from diphtheria during childhood. Malaria, typhoid fever, scarlet fever, measles, influenza and acute rheumatism are responsible for some cases, but to a much less degree than the first mentioned infection. In marantic conditions and as a result of the cachexia of tuberculosis it may be encountered. Toxic cases, notably those seen in adults resulting from alcohol, arsenic and mercury are rare in childhood. There is a class of *idiopathic* cases which are difficult to explain. They are usually described as *rheumatic* and follow exposure to cold or from over-exertion. There is an epidemic type which is in all likelihood an atypical form of poliomyelitis.

The lesions are a degenerative process in the axis-cylinders, not, however, affecting the nerve trunk uniformly and completely. This is associated with hyperemia of the peri- and endoneurium. In some of the severe cases of diphtheritic paralysis degenerative lesions have been demonstrated in the cord and even in the brain in association with the neuritis.

Symptoms.—The clinical course of diphtheritic paralysis has been described under *diphtheria*. In non-diphtheritic cases

there is first noticed a general weakness of the muscles, together with pain and tenderness along the affected nerves. Tingling and formication are also frequently complained of. The paralysis which results is usually of wide distribution, producing foot-drop and wrist-drop, inability to walk and spinal curvature. Partial anesthesia likewise develops, and considerable atrophy of the paralyzed muscles may set in. The knee-jerk is abolished, and if power of locomotion is not entirely lost the child shows marked ataxia in walking and standing. In the course of a few weeks improvement sets in, and after a time complete recovery is the rule, although some atrophy and loss of function may persist. Permanent disability is rare in children and the prognosis is good, as the etiological factors responsible for the unfavorable outcome in adults—such as alcohol—do not enter here. A fatal termination may take place in diphtheritic paralysis, or in other cases of rapid onset and wide distribution, in which the respiratory and cardiac innervation becomes involved.

Diagnosis.—The gradual onset, usually during the period of convalescence from an infectious disease or after exposure to damp and cold (rheumatic cases); the symmetrical distribution, and the accompanying sensory disturbances, will differentiate multiple neuritis from *poliomyelitis anterior*, as well as from the various ataxias. Its tendency to progressive improvement and recovery is another feature of diagnostic importance. The presence of pain is an important symptom, especially tenderness along the nerve trunks.

Treatment.—The child should be kept in bed and put on a low protein diet. The affected limbs may be wrapped in cotton and heat should be applied for the relief of pain when this is present. Mild galvanization of the affected nerves and, as atrophy sets in, massage of the muscles are of great benefit. To overcome deformity in the extremities it may be necessary to resort to mechanical devices.

Aconite.—Recent cases following exposure. Tingling and

formication in the affected parts is the chief indication. This and *rhus tox.* are the chief remedies in idiopathic neuritis.

Arsen.—Malarial or cachectic cases; burning pains, general prostration. Marantic origin; cachexia.

Argentum nitr.—Ataxic symptoms.

Causticum is a most useful remedy for localized paralysis due to neuritis, or for the later changes of multiple neuritis.

Gelsemium is useful in the early period of infectious cases, notably in diphtheritic paralysis.

Rhus tox. is of great value in rheumatic cases. Traumatic cases call for *arnica* and *hypericum*, especially the latter.

HEADACHE.

A variety of conditions, notably anemia, chlorosis, lithemia, eye-strain, neurasthenia, hysteria, constipation and gastric derangements may give rise to headache. In inflammatory and organic brain affections it is a prominent symptom, and in the infectious fevers and in uremia it is quite constantly present. Syphilitic headache is rarely encountered in children. Headache is less common in children than in adults and is, as a rule, of more serious import especially when it is associated with fever or persistent in character. Under these conditions we should always consider the possibility of typhoid fever or meningitis and in the persistent, non-febrile type an intracranial condition should be suspected.

Migraine is an essential headache, occurring paroxysmally and resulting from nervous discharges in the cortical sensory centres. The *exciting causes* may be any of the disturbances capable of producing headache, such as mental or physical fatigue, eye-strain, constipation, etc. The condition itself is usually hereditary, and is one of the manifestations of a neuropathic constitution, being, so to speak, a sensory epilepsy. In some instances a history of cyclic vomiting is obtained.

The *symptoms* of migraine in childhood are the same as those observed in adults, with the exception that they are not

quite so severe and usually of less frequent occurrence. Scintillating scotomata are often observed, being described as fiery flashes of figures before the eyes. The pain may be confined to one side of the head, and is accompanied by nausea and vomiting, the latter giving relief, as a rule, although indigestion has nothing to do with these attacks excepting that it may act as an exciting cause. Other disturbances—e.g., amblyopia; hemianopsia; aphasia; numbness and tingling in various parts of the body, followed by anesthesia, and possibly paralysis—may be observed during an attack.

The *diagnosis* of migraine is based upon the paroxysmal nature of the attacks, the presence of nausea and vomiting without gastric derangement, and the accompanying sensory disturbances. *Symptomatic headaches* are recognized by their transitory nature and the presence of one of the etiological factors enumerated above. It is important both from the standpoint of prognosis and treatment to exclude *intracranial disease* in these cases and the patient should be observed over a sufficient length of time to determine the true nature of the case.

Treatment.—Children subject to migraine should be carefully dieted, especially avoiding the excessive eating of carbohydrate foods, particularly sugar. Rich foods must be excluded from the diet and such articles as chocolate, ice-cream, candy, cakes and nuts are especially harmful. Strict attention to the bowels is necessary although there may be no apparent constipation. Errors of refraction must receive prompt attention.

The most useful remedies in headache and migraine are *iris*, *nux vomica* and *sanguinaria*. *Iris* is indicated in "bilious" headaches accompanied by a blur before the eyes and vomiting of bile. *Nux vomica* is especially helpful in cases traceable to dietetic indiscretions with coated tongue and constipation. *Sanguinaria*, is indicated in the neurotic type of migraine and should be given between the attacks as well as during the same.

Bell.—Congestive headache; throbbing of the carotids;

throbbing pains in the temples; face flushed. The pain is worse lying down, and is temporarily relieved from sitting up and by binding the head up tight.

Cham.—Beginning with flickering and fiery zigzags before the eyes. Great irritability of temper. Neurotic cases.

Gelsemium.—Purely neuralgic type of headache.

Ignatia.—Hysterical headache; *clavus hystericus*; from emotional excitement or overpressure at school. Highly nervous temperaments.

Iris.—The attack begins with dimness of vision and terminates with the vomiting of a yellowish, bitter, sour-smelling fluid. Usually right-sided.

Nux vomica.—Headache traceable to errors in diet and neglect of the bowels. Nervous, excitable temperament, awakening in the morning with headache.

Sanguinaria.—Pain beginning in occiput and spreading over the top of the head, settling over the right eye. Great sensitiveness to light; flushes of heat and alternate chilliness. The attack ends in vomiting. Headache in chlorotic subjects. Subdued, tearful temperament; anorexia with coated tongue, no thirst.

CHAPTER XV.

DISEASES OF THE EAR, NOSE, AND THROAT.

OTITIS.

Inflammation of the middle ear is of common occurrence during infancy and childhood, although it is a condition that is frequently overlooked unless an ear discharge appears. Failure to make an early diagnosis in a case of purulent otitis media and neglect to drain the eardrum may result in the development of mastoiditis and permanent impairment of the hearing. Every case of otitis, however, does not present so serious a prognosis. As will be seen from a description of the affection, there is a mild catarrhal form complicating rhinopharyngitis or apparently occurring primarily and a serious suppurative variety which is usually a complication of one of the acute infectious diseases. Cases complicating scarlet fever are noted for their severe course.

The external auditory canal is directed more forward in the infant than in the adult, for which reason it is at times necessary to draw the lobe of the ear downward and forward in order to insert the speculum instead of drawing the aurical upward and backward, as in adults. The Eustachian tube is wider, shorter and more horizontally placed than in the adult, and this anatomical feature, in conjunction with the prone position so constantly assumed by infants, offers the explanation why extension of an infection of the nose and throat travels so readily to the tympanum. The tympanic orifice is larger than the pharyngeal. Inflation of the middle ear is more easily accomplished than in adults.

The membrana tympani, or drum head, is almost horizontally placed, gradually assuming the perpendicular position as the ear develops. It is thicker than in the adult and does not rupture so readily spontaneously.

The tympanic cavity is bounded superiorly by a thin plate of bone upon which the middle lobe of the brain rests. In the infant a suture, the petroso-squamosal, is found, allowing a vascular communication between the middle ear and the dura mater. For this reason meningeal irritation is so commonly observed in conjunction with otitis media. The close proximity of the inferior wall to the jugular fossa accounts for the tendency to phlebitis and thrombosis of the jugular vein as complications.

The upper portion of the tympanic cavity containing the malleus and part of the incus is known as the attic. It communicates with the mastoid antrum, and for this reason an accumulation of pus in the tympanum reaching to or confined to this point is usually followed by infection of the mastoid process. On account of the underdeveloped state of the mastoid, however, involvement of the petrous bone and of the brain is more common than mastoiditis.

The mucous membrane lining the tympanum is quite thin and vascular, presenting a reddish and swollen appearance in young infants.

The mastoid process is but a small, undeveloped tuberosity at birth and contains, as a rule, only one cell, the antrum. It gradually develops by extending downwards and at the age of five years reaches the adult type. The upper wall of the antrum is in close proximity to the dura mater, being separated therefrom by only a thin layer of bone.

The facial nerve passes along the upper portion of the tympanic cavity and downward through the mastoid cells. For this reason it frequently becomes affected in middle ear and mastoid disease.

Earache is the most prominent symptom of otitis, but it is possible for an inflammation of the middle ear to exist without pain. This sometimes occurs in marantic infants, in whom an ear discharge may be the first sign of trouble. Again, the pain may be vague and not definitely localized or be masked by

cerebral irritation, but in these cases pressure at the tragus will usually elicit tenderness.

Tenderness and redness (inflammatory blush) over the mastoid process indicates involvement of the mastoid cells and is an unfavorable symptom.

Discharge.—In the acute forms of otitis media that lead to perforation of the membrana tympani the discharge is at first serous, later becoming muco-purulent. In the severe form, namely, that complicating *scarlet fever*, it is usually purulent from the beginning; the ordinary catarrhal variety, however, may assume a purulent character if its course becomes protracted.

Tuberculosis.—In the tuberculous variety of otitis the mucous membrane of the tympanic cavity is pale and the discharge is watery or a thin pus, in which the tubercle bacillus may be demonstrated. Multiple perforation of the membrana tympani is characteristic of tuberculous otitis.

Influenza.—Otitis media is a frequent complication of influenza and during epidemics of this disease many cases of otitis are usually encountered. In this variety the discharge is at first sero-sanguinolent, later becoming sticky. There is always more or less blood, on account of the great congestion of the mucous membrane of the tympanum and of the drum head.

ACUTE CATARRHAL AND ACUTE PURULENT OTITIS MEDIA.

The two varieties will be considered under the same heading, as it is impossible to draw a sharp line of distinction between them. The catarrhal variety is by far the commoner in infants, while in older children the purulent variety predominates. The explanation of this lies in the fact that catarrhal otitis usually develops secondarily to an acute nasopharyngitis, while the purulent variety develops in the course of one of the infectious diseases, notably, scarlet fever and measles, and less frequently in typhoid fever, pneumonia and diphtheria. Influenza is a common cause of the more severe

catarrhal cases. The micro-organisms most commonly found in the discharge are the pneumococcus and the streptococcus; the latter is responsible for the damage done to the middle ear and adjacent structure in scarlatinal otitis and the other grave symptoms of suppurative otitis. Adenoids are the most prominent predisposing cause.

Symptoms.—In infants otitis is usually preceded by a naso-pharyngitis; as the ear becomes involved there is an increase of fever and earache sets in. Although the child frequently gives evidence of the seat of the pain by putting the hand to the side of the head and by crying when the affected ear is touched, still there are a great many cases in which earache is not suspected until the membrana tympani has ruptured and a discharge makes its appearance. This is especially the case when otitis complicates an acute illness, such as pneumonia, for example. In these cases there will be a persistence of fever that cannot be accounted for and the child will cry incessantly for no known reason. In the course of a day or two the appearance of the ear discharge clears up the mystery. Sudden exacerbation of fever in any acute illness not explained by other complications and persistent crying should always lead to an examination of the ears.

In older children the disease is ushered in with excruciating pain and high elevation of temperature. Pain begins in the ear, but radiates over the entire side of the head. As a rule, it is promptly relieved when perforation of the membrana tympani takes place.

Often the symptoms closely resemble meningitis; the disease is ushered in by convulsions and vomiting, and marked cerebral irritation is present on account of the close connection between the middle ear and the dura mater. These symptoms, however, disappear as soon as the middle ear is evacuated.

Early in the disease the drum head in the region of Shrapnell's membrane is congested. There is also hyperemia extending along the posterior border of the handle of the malleus;

the drumhead loses its lustre and assumes a deep pink color varying with the intensity of the inflammation. The external auditory canal also becomes deeply congested.

At first the drum head is somewhat depressed, but as the exudate fills the tympanic cavity it bulges, especially in its posterior half. When perforation occurs it most frequently takes place in the lower anterior or posterior quadrant of the membrane. Spontaneous perforation is less apt to drain the tympanum as thoroughly as an artificial puncture, nor does it heal as well.

When the pain continues after perforation, we should suspect involvement of the periosteum or of the mastoid cells.

The complications of otitis media are mastoiditis; facial paralysis; meningitis; cerebral abscess; septicemia; thrombosis of the lateral or other sinuses; facial erysipelas and eczema aurium.

Prognosis.—This is admirably summed up by Palen and Clay (*The Practitioner's Otology*, The John C. Winston Co., 1921) as follows: "On account of the complications which may occur during the course of this disease, a guarded prognosis is at all times advisable. The prognosis depends, to a large extent, upon the causal factor and upon the method of treatment. Cases originating from scarlet fever, measles or diphtheria, are especially apt to be virulent and to become chronic. The otitis occurring with certain epidemics of influenza is also frequently of a very virulent character, while those occurring from simple colds, exposure and nasal obstructions are less severe in character and less destructive. The prognosis, in all cases, will depend also upon the type of infecting organism and the resistance of the patient. A virulent organism in a patient with good resistance may produce a mild type of otitis media, while in a patient with markedly lessened resistance it will exhibit its virulency to a marked degree. Among the most virulent aural bacteria are the different types of streptococci and the pneumococci. The prognosis, where these are present, is less favorable, as regards duration of the

condition and tendency to complications, than it is if the staphylococci are the causal bacteria."

Diagnosis.—Earache should always be suspected when an infant cries continuously or when the fever suddenly rises during the course of an acute illness without assignable cause. When enlarged tonsils and adenoids are present the probability of earache should always be born in mind. Inspection of the ear drum will give positive evidence of the disease.

Treatment.—Absolute rest in bed should be enforced and much relief of suffering may be obtained by instilling hot water into the external auditory meatus, or better, by the instillation of a one per cent solution of carbolic acid in glycerin. This procedure is not only useful in relieving the pain, but will at times abort the attack by osmotic action through the membrana tympani, and in any case it will render the canal aseptic in anticipation of perforation, natural or artificial (C. M. Thomas). The use of oily preparations in the ear should be discouraged.

The most important remedies are *aconite*, *belladonna* and *pulsatilla*. Their use is not limited to the homeopathic school. Thus, Bacon (*Manual of Otology*) says: "*Aconite* in drop doses is a most valuable remedy when there is fever and especially in cases due to cold. Tincture of *pulsatilla*, likewise given in drop doses, is indicated also in cases in which there is profuse discharge from the nares or naso-pharynx, and may be administered alternately with *aconite*." The nose and throat should also receive attention. Unless the pain is promptly relieved by these measures and when the fever and appearance of the membrane indicate an accumulation in the middle ear, incision of the drumhead should be performed. Palen and Clay (*loc. cit.*) express themselves as follows on this subject: "While many pages have been written upon the local treatment of acute otitis media and especially upon ways and means to lessen or control the pain occurring in these cases, we believe that the only treatment for the relief of pain, which occurs before the

drum has perforated, is the establishment of good drainage by means of an *early and free incision of the drum.*"

A successful paracentesis is a free incision of the membrane and not merely a puncture. It should be done under a general anesthetic. The technique is as follows: the patient having been anesthetized and the external auditory canal thoroughly cleansed with a hot 1-5000 bichloride of mercury solution, the drumhead is inspected with the aid of a speculum and head mirror in order to determine the site of bulging if this be demonstrable. The incision is made with a narrow bistoury or tenotome. Ordinarily the line of incision extends from just behind the stapes to the lower border of the drumhead, closely hugging the bony structure of the posterior canal. In grave cases, with bulging of the drumhead in its posterior and upper quadrant, together with indications of mastoiditis, the incision should be carried well up the posterior fold and into the attic. At the same time the knife should be brought out along the upper posterior wall of the external auditory canal to relieve all tension. The canal is then lightly packed with sterile gauze and after the acute symptoms have subsided irrigation with a saturated solution of boric acid may be resorted to several times daily. Inflation, cautiously employed, when the perforation is large, helps to remove the secretion from the tympanum.

Remedies.—In the acute stage, *aconite* and *pulsatilla* are most commonly indicated (see above). *Belladonna* is the remedy when cerebral symptoms are prominent. *Capsicum* is highly recommended for the early stages of mastoid involvement.

During the period of discharge, *pulsatilla* and *calcareia iodid.* are most useful. *Hydrastis* is particularly indicated in influenzal cases, when the discharge is sticky and tenacious. When the discharge excoriates we should think of *mercurius* and in involvement of the bone *silica* is the most useful remedy.

ACUTE TONSILLITIS.

Acute inflammation of the tonsils may be either superficial, or catarrhal; follicular, or cryptic; and parenchymatous. Anatomically the tonsils consist of an aggregation of lymphoid tissue embedded in connective tissue and covered by a mucous membrane from whose surface numerous mucous glands dip into its parenchyma. These glands form so-called crypts, or follicles, and they play an important role in the diseases of the tonsil.

Clinically the tonsil is an important port of entrance of the organisms of many of the infectious diseases. Diphtheria and scarlet fever primarily attack the tonsils, and rheumatic fever can frequently be traced to a tonsillar infection.

Acute Superficial Tonsillitis.—As the name implies, acute superficial tonsillitis involves only the mucous membrane covering the tonsil. The process may also spread to contiguous structures, and it either undergoes prompt resolution or in the case of secondary infection is followed by superficial necrosis of the epithelium, or suppuration of the connective tissue takes place, resulting in peritonsillar abscess.

It is a common accompaniment of many of the infectious diseases notably measles and scarlet fever. In primary cases the usual etiological factor is "taking cold," and by many it is believed that the "rheumatic diathesis" offers especial predisposition to these attacks.

Symptoms.—In primary cases there is malaise and slight chilliness, together with dryness of the throat and some pain on swallowing. The tonsils appear bright red, swollen, and their surface presents a somewhat edematous appearance. It is seldom that the process ends here, however, the crypts usually becoming occluded and filled with fibrin, leucocytes and epithelial debris, which constitutes acute follicular tonsillitis.

Associated symptoms are fever; headache and malaise; stiffness of the neck, even torticollis and earache.

ACUTE FOLLICULAR TONSILLITIS.

Acute follicular, or *cryptic* tonsillitis, is an acute infection of the tonsils. The germs usually found are the streptococcus, staphylococcus, and pneumococcus. As a rule the infection is limited to the crypts of the tonsils whence the name of the disease. In some instances the process extends beyond the mouths of the crypts and spreads over the surface of the tonsils producing a clinical condition simulating diphtheria. These cases are usually due to a streptococcus infection and may occur as epidemics of "*septic sore-throat*" or as a complication of scarlet-fever (*pseudo diphtheria*).

Symptoms.—The attack begins with malaise and chilly sensations, usually along the spine, followed by fever and aching throughout the body. There is dryness of the throat and some pain on swallowing, but frequently the child does not refer to its throat until the tonsils are greatly swollen. The sudden onset of a chill and high fever without the complaint of sore throat may prove very misleading unless a routine examination of the throat in all acute illnesses is made.

Fever persists for about three days, together with an increment in the severity of the symptoms, ranging between 100° F. to 105° F. By this time the inflammation of the tonsils has reached its climax and they present a characteristic appearance. They are deeply congested, uniformly swollen and their surface is studded with yellowish-white, punctate spots appearing at the mouths of the crypts. When the exudation is abundant it spreads over the surface of the tonsils and may give rise to the appearance of a membrane. This is, however, readily wiped off. Again, necrosis of the epithelium around the mouths of the crypts may occur, the spots assuming an irregular outline, like a diphtheritic membrane, and these spots may coalesce; but the deposit is only superficial and is readily wiped off, distinguishing it from diphtheria.

The lymphatic glands of the neck may become enlarged and tender, but never to the extent found in diphtheria.

Associated symptoms are painful deglutition; lancinating pains extending into the ears; headache and prostration.

The tongue is coated and slimy; the breath is offensive, and there is anorexia and constipation.

The fever subsides on about the third day; the tonsillar swelling abates at the same time, and convalescence is established in the course of a few days.

Diagnosis.—The most important condition from which follicular tonsillitis is to be distinguished is diphtheria. In a typical case this is comparatively easy, but in the class of cases described as pseudo-diphtheria many difficulties are encountered.

The characteristic points to be remembered in the diagnosis of follicular tonsillitis are: the punctate spots of soft, unorganized exudation confined to the tonsillar crypts; the uniform inflammation and swelling of the tonsils; the bilateral character of the affection; the high fever and pain and the absence of profound toxemia; and, lastly, the absence of marked enlargement of the lymphatics of the neck. In all doubtful cases, however, a bacteriological examination of the exudate should be made.

Treatment.—The child should be put to bed and isolation of the patient enforced. When there is much pain and swelling of the tonsils an ice collar will give relief. The throat may be sprayed several times daily with a mild antiseptic, such as boric acid or hydrogen peroxid. Local applications of a ten per cent solution of argyrol may also be employed.

The most important remedies are: *belladonna*, *mercurius iod. rubr.* and *apis*.

Belladonna is indicated in the early stage when there is dryness and redness of the throat with pain on swallowing; throbbing headache; photophobia; high fever and flushed face. It is more frequently indicated in tonsillitis in children than in adults.

Apis is indicated when edematous swelling of the mucous membrane is the leading feature in the case. There are sharp, sticking pains on swallowing.

Mercurius iod. rubr. is the most useful remedy in the fully developed stage of the affection.

VINCENT'S ANGINA.

Vincent's Angina, or *ulcero-membranous tonsillitis*, is an affection bearing a superficial resemblance to diphtheria but presenting none of the toxic manifestations of this disease. In *ulcero-membranous tonsillitis* the tonsil becomes covered with a dirty-yellowish exudate; this is often confined to a single tonsil. When the exudate is wiped away, especially if done roughly, a bleeding surface may remain. The lymphatics at the angle of the jaw on the affected side are swollen. Thus far there is a strong resemblance to diphtheria, even to offensive breath, but constitutional symptoms are slight or wanting and a bacteriological examination reveals instead of the Klebs-Loeffler bacillus the fusiform bacillus of Vincent and its accompanying spirillum. Ulcerative stomatitis may be an associated condition.

The *treatment* is the same as for other forms of tonsillitis. Locally, *hydrogen dioxid*, preferably as a spray, is the most useful cleansing agent. It may be followed by the application of *iodine* and *glycerine* (one part tincture of iodine to five parts glycerine).

Merc. iod. rubr.—This is the most useful remedy in cases resembling diphtheria where there is superficial ulceration of the tonsils; fibrinous exudation and enlargement of the cervical lymphatics.

ACUTE PARENCHYMATOUS TONSILLITIS; PERITONSILLAR ABSCESS.

Acute parenchymatous tonsillitis, commonly called "quinsy," results from an infection of the peritonsillar tissue. Suppura-

tion as a rule sets in, taking place in the peritonsillar connective tissue and terminating in the formation of an abscess which may rupture into the pharynx either anteriorly or posteriorly, following the line of least resistance. It is a disease common in later childhood and in adolescents.

Symptoms.—The onset is similar to that of other forms of tonsillitis, with the exception that the inflammation is one-sided and attended with more pain and swelling. The pain at first is lancinating; later it becomes throbbing in character. There is a constant desire to swallow, which adds greatly to the discomfort of the patient. Fever and malaise are usually not so marked as in follicular tonsillitis.

On inspection, the throat presents a swollen, edematous appearance and a tumefaction arising from the tonsillar region is seen projecting toward the median line. The tonsils and pharynx are covered with a grayish, viscid mucus which gives the appearance of a thin pseudo-membrane, but by spraying the throat it can be completely removed. The tonsil itself is not the seat of the chief swelling, but it is carried into the median line by the surrounding tumefied structures. The opposite side may become affected later on, but the disease is rarely bilateral. Inspection is difficult on account of the stiffness of the jaw that is associated. Fluctuation may be elicited, but it is not always easy to determine on account of the boggy, edematous condition of the tissues.

The duration is from a few days to a week or longer. Resolution may set in, or spontaneous evacuation take place after four or five days with prompt relief of the symptoms.

Treatment.—If suppuration cannot be aborted by the use of the ice-bag and the indicated remedy, the abscess should be evacuated as soon as pus is suspected and an antiseptic gargle freely used. The incision is made with a sharp pointed bistoury whose cutting edge has been wrapped in cotton, exposing only the point for a distance of about a quarter of an inch. The point is inserted to its full length into the substance

of the half arch just above the tonsil and a quarter of an inch from its free border. Peritonsillar abscess can often be most satisfactorily evacuated by passing a bent probe outward and upward posteriorly to the anterior half and into the supra tonsillar fossa (Thomas). The patient should then gargle with a warm 2 per cent *boric acid* solution, or diluted *hydrogen dioxid* so long as pus is present.

Remedies.—*Belladonna* in the early stage; later as soon as pus begins to form, *mercurius vivus*; and *hepar sulph.*

Apis may become indicated from a predominance or edema.

Capsicum.—Serous infiltration of the faucial tissues; boggy, not edematous, in appearance; left side worse; pain burning, stinging. When tongue is heavily coated white, uvula edematous, especially with a dusky infiltration of the left pillars and some swelling of the lymphatic glands, *caps.*, in the 3x or 6x, will usually relieve inside of twenty-four hours (Ivins).

Phytolacca.—Chills and fever alternate; prostration; pain running to ears on deglutition; affected parts dark-purple, almost blue; rheumatic subjects; uvula enlarged and edematous.

Silicea.—Protracted cases. Suppuration continues after evacuation of pus has taken place (*calc. sulph.*).

HYPERTROPHY OF THE TONSILS.

There are two varieties of hypertrophy of the tonsils; in the one the increase in structure is mainly glandular, while in the other it is interstitial. The first variety is known as the soft, glandular type; the other as the hard, fibroid, or lobulated tonsil. An enlarged tonsil is not necessarily an hypertrophied one, as enlargement may result from vascular engorgement and does not necessarily indicate cell proliferation. Again, in children the tonsils are normally large, and because they extend beyond the pillars of the fauces, it does not necessarily follow that they are hypertrophied (Kyle). At the time of the eruption of the last molar teeth a physiological

enlargement of the tonsils occurs which may subside as the child grows older.

The *cause* of the various enlargements is both constitutional and acquired. The scrofulous diathesis and the status lymphaticus are constitutional states predisposing to hyperplasia of all lymphoid structures. Recurring attacks of tonsillitis lead to hypertrophy of the tonsils and all infectious diseases which attack the tonsils aggravate the same. In many cases no etiological factor can be discovered. Often enlarged tonsils and adenoids appear as a family trait.

Symptoms.—Subjective symptoms depend largely upon the size of the tonsils. They may be so large as to cause considerable interference with normal respiration by filling up the pharyngeal space, and under these circumstances the voice is also affected, acquiring a nasal twang. Many of the symptoms resulting from adenoid vegetations are also caused by enlarged tonsils. Interference with the appetite is a common disturbance and when the tonsils are infected the manifestations of a focal infection can usually be elicited. An infected tonsil is not necessarily an enlarged tonsil. It may be partly submerged in the tonsillar fossa or covered by the plica triangularis and appear as a small tonsil when in reality it is hypertrophic and diseased. (Palen and Clay.)

In the soft variety the tonsil is uniformly enlarged, while in the fibrous variety it is lobulated; the crypts are abnormally large, and its consistency is hard and unyielding.

Treatment.—Unless the tonsils are sufficiently enlarged to interfere with the child's health, or to affect the voice, they will require no further treatment than mild local measures and a remedy prescribed upon a constitutional basis. It is the simple, hypertrophic variety of enlarged tonsil without connective tissue proliferation that so promptly improves under appropriate treatment and undergoes physiological atrophy in later life. The fibroid variety, however, is rarely improved by treatment of any kind, and if it be large enough to cause symp-

toms it should be enucleated. This also holds good in the case of infected tonsils whether they be large or small. If there is a history of recurring attacks of tonsillitis and if the sub-tonsillar lymphnodes are enlarged the tonsils are in all probability infected. Should rheumatic symptoms or other evidences of focal infection be present the tonsils should be removed.

In simple hypertrophy of the tonsils *calcareo phos.* has proven clinically useful.

RETRO-PHARYNGEAL ABSCESS.

Retro-pharyngeal abscess results from an acute infection of the lymphatic glands and vessels of the pharyngeal space. A *septic* variety, occurring as a complication of scarlet fever and measles, is sometimes encountered, but it is much rarer than the idiopathic form. *Chronic retro-pharyngeal abscess* is due to cervical Pott's disease. This occurs in childhood, while the above condition occurs almost exclusively during infancy.

As the lymph-nodes of the retro-pharyngeal space are intimately connected with the lymphatics of the tonsils and uvula, any acute inflammatory condition of these structures may result in involvement of the pharyngeal lymphatics. This is especially the case during infancy. Later in childhood, however, these glands undergo atrophy, for which reason retro-pharyngeal suppuration is rare after the third year.

The tumefaction may be situated in the median line, but more frequently it is to one side and may even appear to arise from behind one of the half-arches. The glands at the angle of the jaw may also be implicated, in which case the swelling is found at or beneath the angle of the jaw and in front of the sterno-mastoid muscle. In such cases a spontaneous evacuation of the abscess externally may take place, although the majority break into the pharynx.

Septic retro-pharyngeal abscess complicating scarlet fever and measles shows a tendency to burrow into the mediastinum

or ulcerate into the carotid arteries and other important structures.

Symptoms.—The onset is insidious and usually it is not suspected until marked symptoms have developed. The early symptoms are those of an upper respiratory infection but instead of the fever subsiding in the course of a few days it continues and the symptoms of pharyngeal obstruction develop. There is difficulty of breathing, especially on inspiration; crowing respiration, due to inco-ordination of the vocal cords; retraction of the head in order to give the larynx as much free space as possible and distinctly nasal cry. The child breathes with the mouth open and holds the head so rigid that cervical Pott's disease or meningitis may be suggested. Inspection of the throat will, however, immediately clear away any doubt as to the true nature of the case. The abscess is readily made out by carefully introducing the index finger into the pharynx.

If allowed to rupture spontaneously the pus may be aspirated into the lungs, causing suffocation or setting up a *septic broncho-pneumonia*; it may also find its way into the Eustachean tubes and set up an acute otitis. In many instances, however, the pus is swallowed or evacuated through the mouth without causing any trouble. Nevertheless, prompt surgical interference offers the best prognosis and should be instituted in all cases as soon as they give indications for the evacuation of pus.

Treatment.—The abscess is easily incised when it points to the median line or not far therefrom. Cases in which the swelling is well to the side require greater care, as there is danger of wounding the carotid artery. Those pointing externally require expert surgical attention. Tuberculous abscesses should be opened externally whenever possible.

The child is held firmly in the upright position and the throat illuminated by means of a head-mirror. A mouth-gag is unnecessary; all that is required to expose the abscess and keep the mouth open is a reliable tongue-depressor. The incision is

made toward the median line with a bistoury whose cutting edge has been protected by wrapping it with cotton up to within half an inch from the point. After making the incision it is often necessary to break up septa of connective tissue within the abscess cavity with the tip of the index finger.

The remedies indicated are *belladonna* in the early stage and *hepar sulph.* when pus begins to form.

ACUTE RHINITIS; PSEUDO-MEMBRANOUS RHINITIS.

Acute rhinitis is an acute inflammation of the mucous membrane of the nasal cavities occurring either as a primary condition or secondary to one of the infectious diseases, notably measles and influenza.

Pseudo-membranous rhinitis associated with faucial diphtheria is due to the *Klebs-Loeffler bacillus* in its most virulent form, while in those cases in which a diphtheritic membrane develops primarily in the nose running a mild course, the bacillus is present in attenuated form. Such cases, however, may give rise to a severe faucial diphtheria, and for this reason every case of pseudo-membranous rhinitis should be isolated. This attenuated diphtheria bacillus is known as Von Hoffman's bacillus. According to Park (*Bacteriology in Medicine and Surgery*) only in a few cases have other bacteria been found to cause the croupous exudate; they were mainly the pyogenic cocci.

There is no doubt that rhinitis is mildly contagious. A natural predisposition is found in many cases; this is particularly the case in anemic children that have been reared like hot-house plants and in those suffering from adenoids and enlarged tonsils.

Symptoms.—Following upon exposure, or "catching cold" or in the course of an infectious disease a sense of fulness in the nostrils with dryness of the mucous membrane develops, succeeded by an acrid, watery discharge consisting of serum with a small amount of mucus. At this stage the mucous

membrane appears red and swollen, and the entire nasal cavity may be occluded by the swollen turbinated bodies.

In primary cases a slight febrile reaction sets in and there is headache, and lassitude. Mild cases may be aborted at this stage and resolution occur without any further developments. In infants these attacks are spoken of as *snuffles*, and unless they are due to syphilis or are benign, profuse mucopurulent secretion makes its appearance, flowing freely from the nose and covering over the entire mucous membrane of the nasopharynx. The process may extend to the frontal sinuses, the Eustachian tubes and middle-ear, and to the pharynx. If the infection has been of a virulent nature ulceration of the mucosa and suppuration of the middle-ear are liable to supervene.

Pseudo-membranous rhinitis is almost invariably diphtheritic in origin, as has been stated above. From the fact that constitutional symptoms are usually slight in primary diphtheritic rhinitis, it frequently remains unsuspected until the membrane is accidentally discovered. The membrane may persist for weeks, coming away in large pieces. If during its course it be removed, it usually recurs. The nose is obstructed, and a thin blood-streaked discharge is present. Such a secretion should always arouse suspicion of diphtheria. On inspection, the membrane is seen as a firm, grayish exudate upon the interior of the nose. The clinical course is more benign than that of faucial diphtheria but it should receive the same treatment.

Treatment.—In the early stages the obstruction may be much relieved by spraying or douching the nose with a warm, mild, alkaline, antiseptic solution, such as Dobell's solution, or a normal saline solution, followed by spraying with a bland oil containing camphor or menthol in the proportion of two to four grains to the ounce. Later, as the discharge becomes profuse, frequent cleansing of the nasal passages is imperative. In infants or young children who struggle against the use of the atomizer, a small glass syringe may be employed, injecting into one nostril and allowing the fluid to flow out of the other, the child lying on its side during the operation.

In the early stages *aconite* and *gelsemium* are the most important remedies. Hughes (*Manual of Therapeutics*) considers *camphor* a specific in the early stage, promptly aborting most cases and especially relieving the chilly feeling.

Aconite.—Sneezing; fever with restlessness and full pulse; burning of the eyes. Attacks of coryza from exposure to draughts or from being chilled.

Gelsemium differs from *aconite* in the absence of the restlessness and high fever and in the predominance of malaise; chilliness, especially creeps up and down the spine but not a well defined chill; headache with drowsiness and heaviness of the eyelids; aching in the muscles. *Gelsemium* is admirably suited to the ordinary form of grippe.

Nux vomica is indicated in the early stages of many cases; there is dryness and obstruction of the nose; fulness at the root of the nose and frontal headache; cold hands and feet with a hot head; anorexia and constipation; irritability of temper and feverishness. Subjects who are overly sensitive to draughts. In this respect *arsenicum* is similar. "Persons who are rarely without a cold" (Ivins). Sneezing; profuse, watery, excoriating discharge; tendency of cold to travel down the chest.

Belladonna has always been a most satisfactory remedy in my hands for the vascular engorgement of the turbinated bodies. The mucous membrane appears dry and bright red and the nose is much obstructed.

Cepa.—Profuse, acrid watery discharge with lachrymation.

Euphrasia has a profuse nasal discharge which is bland, but an excoriating lachrymal discharge, the opposite condition of *cepa*.

Sanguinaria canadensis or *sanguinaria nitr.*, 3x trit., is useful when there is a sensation of great dryness and burning in the nose and pharynx, with headache and loss of smell and taste.

In the second stage, when the discharge becomes profuse and muco-purulent in character, no remedy is more useful in the

majority of cases than *pulsatilla*. When there is much soreness of the nose and evidence of ulceration *mercurius* is the better indicated remedy.

SIMPLE CHRONIC RHINITIS AND PURULENT RHINITIS.

Chronic rhinitis without pronounced hypertrophic or atrophic changes in the nasal mucous membrane is a common affection of childhood.

In the *etiology* recurrent attacks play an important role. The period of childhood itself predisposes to catarrhal inflammations. Children of the scrofulous diathesis and infants presenting symptoms of the exudative diathesis are especially prone to purulent rhinitis. As a predisposing cause, adenoids undoubtedly play a most important role. Unhygienic surroundings, and want of attention during acute attacks or failure to guard against the recurrence of such attacks are also responsible for many cases. No specific micro-organism is present, but there is no doubt that an infection of a mixed character causes the purulent inflammation. Irritation by foreign bodies or other sources of irritation may induce similar pathological changes.

Symptoms.—The chief symptom is a profuse muco-purulent discharge. Nasal obstruction is not pronounced. The nose may become reddened about the orifice and excoriated and crusts form in the anterior nares, usually at night, in this way inducing mouth breathing during sleep. Susceptibility to acute attacks seems lessened on account of reduced sensibility of the mucosa from loss of epithelial cilia (Ivins).

Atrophic changes will occur in the course of years if the progress be not arrested. It may also pass into the hypertrophic variety if rhinorrhea has not been a prominent feature of the case. In scrofulous children infection of the cervical lymphatics is a frequent complication. In the majority of cases the prognosis is good, especially under proper treatment.

HYPERTROPHIC RHINITIS; ATROPHIC RHINITIS.

Hypertrophic rhinitis is a chronic catarrhal inflammation of the nasal mucosa and sub-mucosa, characterized by hypertrophy of the turbinated bodies with resulting nasal obstruction. It is not as frequently encountered in children as in adults, nor is it as common a disease as atrophic rhinitis. The pathological changes require a long time for their development, being a hyperplasia of the cellular elements and overgrowth of the connective tissue and blood-vessels that form the turbinated bodies.

A variety of hypertrophic rhinitis in which there is simply engorgement and dilatation of the blood-vessels is not uncommon. In this class a complete temporary retraction of the mucous membrane may be induced by the local application of cocaine.

Atrophic rhinitis, or *ozena*, is characterized by atrophy of the mucous membrane, of the cavernous structures, and the underlying bone. There is also atrophy of the mucous glands with consequent impaired function and the formation of offensive crusts. The crusts represent inspissated muco-purulent secretion which accumulates in the nasal chambers and undergoes decomposition. They are the cause of the fetor emanating from these patients.

Etiology.—Adenoid vegetations play an important role in the etiology of *hypertrophic rhinitis*, by interfering with the drainage of the nasal chambers, thus inviting the accumulation of irritating material which keeps up a constant congestion of the mucous membrane. Again, the constitutional peculiarity which invites adenoids and hypertrophy of the tonsils predisposes to chronic catarrh and hypertrophy of the intra-nasal structures. Clinically there is an intimate association of these conditions. Another cause will be found in recurrent acute attacks which may lead up to permanent structural changes.

Atrophic rhinitis may develop as an independent affection or

as a sequel to hypertrophic rhinitis. Casselberry dissents from the latter view, believing the transition of an hypertrophic rhinitis an exceedingly rare, and in all events slow process; and he looks upon atrophic rhinitis, particularly in children, as a distinct affection. A pronounced hereditary predisposition, moreover, has often been observed. Bosworth believes suppurative rhinitis of children to be the cause of atrophic rhinitis, the suppurative process destroying the mucosa layer by layer in the course of time, until eventually the deepest structures become involved.

Symptoms.—The chief symptom of *hypertrophic rhinitis* is nasal obstruction. This may be more or less complete and involve both sides simultaneously or alternately. Remissions occur, and frequently the nose will be clear under ordinary circumstances, only clogging up when irritated by the inhalation of dust; walking in the wind; entering a warm room, etc. This peculiar behavior readily explains itself when we remember that the obstruction depends upon the degree of vascular engorgement present at the time.

As a result of the reflex irritation in the nose and the interference with respiration, a train of symptoms indicating a disturbance in the general health of the child arises. Nervous irritability; disturbed sleep and mouth breathing; intellectual torpor; hemicrania; spasm of glottis; asthma and enuresis, all may have their origin in the nasal stenosis.

On inspecting the anterior nares we will find the turbinated bodies swollen and of a deep red color, the inferior turbinated being most readily seen and darker in color than the middle or superior. If there be much engorgement it will be impossible to see more than the inferior body and at the most the anterior half of the middle body without making an application of cocaine to shrink the mucous membrane. Polypi are likely to be confounded with an hypertrophied turbinated body, but they are paler in color, are movable, and occupy a position between the turbinated bodies.

Atrophic rhinitis is characterized by the formation of crusts and fetor. Obstruction of the nares only occurs if the crusts are allowed to accumulate in large masses. They may occur simply as scales, or form in large horny masses, completely occluding the nasal chamber. These masses eventually soften by decomposition or cause necrosis of the underlying mucous membrane, coming away in large masses and leaving an ulcerated surface behind. The fetor may be so intense as to render the patient's proximity unbearable. In the beginning the child may be annoyed by the odor, but eventually the sense of smell becomes so obtunded that it is not aware of the fetor. There may be a sense of distressing fulness in the nose when crusts accumulate, and the habit of constantly picking the nose is soon acquired. Epistaxis is soon a frequent accompaniment. The general health is naturally affected; hearing becomes impaired, and the sense of smell may be entirely lost.

Inspection reveals a spacious nasal cavity lined with a thin, smooth mucous membrane, covered with crusts. Its surface is studded with superficial ulcers. *Hereditary syphilis* is to be differentiated from atrophic rhinitis; in the former there is not a uniform distribution of the atrophic process, and there is deep ulceration and cicatrization. Perforation of the septum with sinking in of the nose is pathognomonic of syphilis.

The *prognosis* is not unfavorable. Under persistent treatment most cases in children recover, some in the course of a few months, others not yielding to treatment in less than a year or two. Syphilitic cases, if seen early before destructive changes have set in, respond promptly to appropriate local measures in conjunction with anti-syphilitic remedies.

TREATMENT OF CHRONIC RHINITIS.

In undertaking the treatment of a case of hypertrophic rhinitis we must first of all determine whether it is an independent affection or due to adenoid vegetations. If adenoids are present they must be removed before the rhinitis can be

benefited. If the condition has not advanced beyond the stage of vascular engorgement a cure usually ensues upon the removal of the adenoids. When permanent hypertrophy of the turbinated bodies has set in surgical treatment is indicated.

Milder cases, not requiring surgical interference, should receive local applications of *iodine* and *glycerine* (5 per cent.), made by means of absorbent cotton on a probe, about twice weekly, followed by the use of an oily spray.

In treating *atrophic rhinitis* the most rigorous steps for maintaining absolute nasal cleanliness must be taken. The free use of the douche bag is here to be instituted, and a pint of Dobell's solution should be allowed to flow through the nares at a time. This should be done twice daily.

If hard crusts have formed that cannot be dislodged by means of the douche, *hydrogen dioxid*, diluted with warm water should be slowly injected into the nares with a blunt syringe; this so loosens them that they can be readily blown out. After the nose has been cleared a few drops of refined carbon oil with iodine (one grain to the ounce) could be dropped into each nostril with a medicine dropper (Kyle).

When eroded surfaces remain after the removal of the crusts a stimulating powder, such as *aristol*, should be insufflated. Syphilitic ulcerations are best controlled by the local application of a ten per cent solution of *nitrate of silver*.

Remedies.—When well marked constitutional indications are present such remedies as *calc. phos.*, *calc. carb.* the *iodines*, *hepar* and *silicea* will give better results than remedies selected purely on local indications. *Pulsatilla* and *hydrastis* are especially useful in *simple*, *chronic* and *purulent* rhinitis.

In *atrophic rhinitis* the *chloride of gold*, *kali bichromicum*, *mercurius corr.* and *silicea* are the most important remedies, *aurum* heading the list.

Syphilitic affections require *mercury*, preferably the *yellow iodide* when the ulceration is confined to the mucous membrane. Ulceration of the septum calls for *kali bichromicum*.

Gummatous infiltration of the soft structures will require the *iodide of potash* in material doses, five grains three times daily being the usual dose necessary.

Alumina.—Thick, greenish-yellow nasal discharge; anosmia; mind sluggish; snapping in the ears when swallowing.

Arsenicum iod.—Delicate tuberculous constitution; acrid discharge with burning in nose. Chronic purulent rhinitis.

Aurum.—Offensive discharge; soreness of bones of nose.

Calc. carb.—Glistening redness of nasal mucosa; extreme sensitiveness of nose; purulent discharge. Chronic purulent rhinitis in scrofulous individuals.

Calc. phos.—Chronic hypertrophic rhinitis in anemic children or in association with enlarged tonsils and adenoids.

Hepar.—Chronic purulent rhinitis with enlarged cervical glands. Hypersensitive to draughts. Uncovering the body brings on attacks of sneezing.

Hydrastis.—Simple chronic rhinitis and purulent rhinitis. Abundant muco-purulent secretion with superficial ulceration of the mucous membrane. The discharge may also be stringy and tenacious. Post nasal dropping.

Kali bichromicum.—Tenacious, yellow secretion; ulceration of the spetum.

Natrum mur.—Simple chronic rhinitis. "In all absence of clear indications for other drugs this is one of the best remedies where persons draw mucus from the posterior nares in the morning."—(Ivins.)

Pulsatilla.—Chronic purulent rhinitis. Profuse discharge which is a bland, thick, yellow muco-pus, streaked at times with green. There is loss of taste and smell, and in order to act well there must be, according to Ivins, the typical *pulsatilla* temperament.

Silicea.—Ozena. Painful dryness of the nose; ulceration with acrid, corroding discharge (*merc. sol.*). Thick, fetid, post nasal discharge. Periostitis. The *silicea* patient is pale and delicate; predisposed to affections of the glands and bones

that undergo rapid destruction; in other words, it presents the tuberculous type. There is also nervous hyperesthesia and tendency to neurotic affections.

ADENOID VEGETATIONS OF THE NASO-PHARYNX.

The muco-lymphoid glands found in the vault of the pharynx and aggregated into a tonsil-like organ known as the *tonsil of Lushka*, or the pharyngeal tonsil, are in their normal state of insufficient size to be readily demonstrated, or to cause interference with free nasal respiration. Under certain conditions, however, they become much enlarged; in some instances a hypertrophy of such extent takes place that they fill up the entire naso-pharyngeal space, thus effectually preventing nasal respiration and giving rise to the pernicious habit of mouth breathing.

No definite cause can be blamed for the development of this hypertrophic condition, as it is encountered in children of all descriptions, although the so-called scrofulous diathesis, and the status lymphaticus are the most frequent constitutional peculiarities found associated with hypertrophied adenoids. During childhood the lymphatic structures throughout the entire body are in a state of active growth and function and there is a normal tendency for lymphoid tissue to become hyperplastic at this age. As in the case of enlarged tonsils, there is a distinct family tendency to hypertrophied adenoid tissue and although the two conditions usually coexist, still it is not uncommon to see children with adenoids whose tonsils are small or rudimentary.

Chronic nasal catarrh; deflections of the septum; the exanthemata, and a damp, changeable climate furnish the causes which excite the hypertrophy of the adenoid tissue.

The *pathological changes* encountered in the mucous membrane of the pharynx are an overgrowth of the muco-lymphoid follicles and of the connective tissue in which they are embedded, together with increased vascularity and thickening of

the mucosa. This hypertrophy leads to the formation of a large glandular mass which may attain sufficient size to entirely block the naso-pharynx. According to the amount of connective tissue present and the mode of proliferation of the glandular elements, there will be either a soft, papillomatous growth, or a hard, smooth mass, known as the individual variety, in contradistinction to the papillomatous, which is a multiple, pear-shaped mass. The individual variety is smooth and firm, while the papillomatous is soft and irregular in contour, conveying the impression of a bunch of earth worms to the examining finger.

Adenoid vegetation belongs practically to the period of childhood, and after full maturity a physiological atrophy as a rule sets in, the pharyngeal vault being usually smooth in adults.

Symptoms.—Chronic nasal and pharyngeal catarrh is usually associated with adenoid vegetations, especially when they have existed for a long time. While a catarrhal affection of the nose and pharynx no doubt often acts as the exciting cause of adenoid tissue proliferation, still adenoids in themselves will set up catarrh through their mechanical interference with the circulation and normal breathing. The obstruction of the naso-pharynx leads to lack of development of the frontal, sphenoidal, maxillary and ethmoidal sinuses with consequent narrowing of the face and upper jaw, which, together with the increased atmospheric pressure exerted upon the buccal surface of the palate due to lessened intra-nasal air-pressure and mouth breathing, leads to a gradual forcing up of the arch of the palate. This deformity results in turn in deflection of the nasal septum, on account of the upward crowding of the base of the septum. In this manner the nasal obstruction is still further augmented and hypertrophic rhinitis is invited.

Deafness from direct pressure upon the ostia of the Eustachian tubes or through an extension of the catarrhal process into the tubes is a frequent symptom accompanying adenoids.

The physiognomy is characteristic and practically pathognomonic, and taken in conjunction with the alteration in voice and impaired hearing makes the diagnosis possible on these data alone. The upper lip becomes shortened from lack of development as a result of keeping the mouth open; the expression of the face is vacant and stupid; the nose is pinched and undeveloped and owing to the contraction of the superior maxilla the permanent teeth become irregular in distribution.

When the condition has arrived at this stage there results as a natural consequence of the interference with the proper aeration of the blood a serious disturbance in the child's general health. Anemia, flat chest and constant colds make the child neurasthenic and may predispose it to pulmonary tuberculosis. The lack of normal physical vigor and the impaired hearing make the child backward at school and frequently stamp it as being mentally sub-normal.

A stubborn nocturnal cough is frequently present. Restless sleep, snoring, night-terrors and enuresis all suggest adenoids. Recurring attacks of earache is another common symptom.

Diagnosis.—The presumptive evidence of adenoid vegetations is found in the facies and the nasal, non-resonant voice together with the associated symptoms of mouth breathing; naso-pharyngeal catarrh; partial or total deafness and retarded nutrition. Naturally these symptoms are only to be encountered in well-advanced cases; in incipient cases the age of the child and the development of the nasal obstructions, not springing from an abnormal condition of the nose proper, should always arouse a suspicion of adenoid vegetations. The positive evidence of adenoids is obtained through posterior rhinoscopy. This procedure is quite difficult, practically impossible with some children. In others, however, a very satisfactory view of the vault of the pharynx may be obtained. Anterior rhinoscopy is usually the most satisfactory method of examination. The inferior turbinated body should be shrunk by the application of a weak solution of cocaine with adrenalin after which the

posterior nares can be inspected. The adenoid tissue is seen in the post-nasal space as an irregular mass lighter in color than the surrounding mucous membrane and when the child swallows or is made to say "e" it moves upward with the elevation of the palate.

Digital examination is a crude and not altogether satisfactory method and should only be used as a last resort.

The *treatment* of adenoids is purely surgical. While conservative measures may be tried in the case of enlarged tonsils, there is nothing to be gained by postponing the removal of adenoid vegetations. Adenectomy is therefore indicated as soon as the presence of adenoids is clinically recognized.

CHAPTER XVI.

DIATHETIC AND CONSTITUTIONAL DISEASES.

MARASMUS, OR ATHREPSIA; MALNUTRITION.

The extreme form of infantile malnutrition, designated marasmus, is a rare condition and is more often seen in hospitals and dispensaries than in private practice. There is a form of malnutrition, however, which is less extreme in type and which is far more common, being a purely dietetic disturbance resulting from improper feeding. Cases of this class are more hopeful and usually respond promptly to treatment in contradistinction to true marasmus in which the prognosis is less favorable.

Marasmus was first described by Parrot in 1877, who designated it "infantile athrepsia." The etiological factors may be a congenital debility or a congenital lack of tolerance for cow's milk and the failure of the child to thrive on artificial food may be noted from the time of birth. In some instances the infant gives a history of having gained weight progressively for a time and then come to a standstill with symptoms of dyspepsia and constipation. A further increase of the food under these circumstances leads to a corresponding loss of weight due to the child's intolerance for the fat of cow's milk. Other cases can be traced to chronic indigestion from overfeeding with carbohydrates, especially sugar. Condensed milk, with its low protein and high sugar content is often responsible for such cases.

The *histological findings* in the gut are not characteristic. Baginsky insists that the mucosa is thinner than normal and that there is distinct evidence of atrophy of the intestinal tubules and villi. Huebner, on the other hand, claims that pathological changes are not constantly found and when so, that they are only the evidence of a preceding enteritis.

On the other hand, the long-continued distention of the gut

with gas, as a result of fermentation accompanied by the wasting of its muscular coat produces the appearance of a glandular atrophy. The careful investigations of Holt substantiate the view that there is no definite gross pathological lesion in the intestinal mucous membrane to account for the clinical manifestations.

The theory of a chronic acid intoxication (acidosis) of intestinal origin was first advanced by Keller, who found the urine highly acid and containing an excess of ammonia. The origin of these acids lies in a deficient oxidation of the carbohydrates and particularly the fats of the ingested food. The excessive elimination of alkaline salts through the intestinal mucosa, which unite with the fatty acids in the intestine, also contributes toward the production of this relative acidosis.

Arguing from the established fact that the intestinal mucosa of a marantic infant assimilates the proteids and fats of an artificial food much less satisfactorily than breast milk and consequently expends a much greater amount of glandular energy in this attempt, Huebner explains the failing nutrition on the grounds of a disturbed balance of energy (Balance Disturbance); in other words, waste of energy on the part of the organism. There is also a loss of mineral salts through the intestinal tract and an increased elimination of nitrogen by the kidneys leading to "Decomposition" (Finkelstein). Loss of fluid and a tendency to dessication is also of great clinical importance in these cases. In a number of cases of severe malnutrition and marasmus I have found a marked deficiency of hydrochloric acid in the gastric contents.

The *etiology* of marasmus is not always clear. In some infants there is undoubtedly a congenital feebleness of constitution which renders them incapable of conquering in the struggle for existence. Here heredity is an important factor, and we may find evidence of constitutional disease in the parents; on the other hand, they may be perfectly healthy. Extreme youth of the mother, and frequent pregnancy at short intervals is

often noted on the material side of the history. The surroundings play an important role. Crowded quarters and lack of fresh air and sunshine are strong contributing factors. The ordinary hospital ward is a most undesirable quarter for infants convalescing from an acute illness and unless promptly removed therefrom they soon show signs of failing nutrition.

Symptoms.—The infant may be delicate at birth, have difficulty in digesting its food even when breast-fed, and its weight curve show a progressive loss of weight interrupted by periods of temporary gain or stand still. More frequently the infant appears normal at birth and gets on perhaps as well as the average one up to the third or sixth month, when, as the result of some acute illness, or what is more common, repeated attacks of indigestion, it loses all tolerance for food and goes into a decline, or “decomposition.” It is by no means necessary that the infant should have been on breast milk and that a change to artificial feeding be instituted in order to bring about this condition. A sudden change during artificial feeding to an ill-selected diet or the more gradual ill-effects from a diet that contains too much sugar or fat will accomplish the same results, especially when the environment is such as to favor marasmus. The emaciation progresses until the infant is reduced literally to skin and bones. The skin is thin and wrinkled and there is a pronounced pallor. Cyanosis is often noted. The face has an old, wrinkled appearance, the eyes being sunken and the small triangular chin showing in marked contrast to the large head; the chest is small and the ribs are plainly visible while the abdomen is flabby and wrinkled or distended. Through the thin abdominal wall the stomach and coils of dilated intestines can often be seen. Impacted fecal matter can often be palpated in the colon. The skin is pale and transparent. There is more or less intertrigo about the genitals and buttocks and a few scattered boils are not uncommon. The urine is ammoniacal and unless great care is taken causes erythema, papules and vesicles on the skin of the buttocks, back and legs.

The temperature is sub-normal and the pulse is weak and slow. A slight fever may be present from indigestion. Edema of the face and extremities may develop without any evidence of a renal complication.

The *stools* vary in character. To all appearances they may be normal, excepting that an excess of fatty acids and calcium soap may be demonstrated. Cases of balance disturbance from fat overfeeding, develop the characteristic large, pale, dry, alkaline soap stools. Cases with sugar dyspepsia have frequent acid stools. Alternate constipation and diarrhea may occur.

The appetite is variable. Sometimes for a considerable period it is voracious and the child does not seem to get satisfied. Then, again, it may be lost and there may be difficulty in inducing the infant to take sufficient nourishment. Troublesome vomiting frequently adds to the gravity of the condition.

The *duration* is difficult to foretell. The child may die from an intercurrent diarrhea or bronchopneumonia, or it may suddenly go into a collapse from which it cannot be resuscitated. On the other hand, progressive improvement may follow under improved hygienic surroundings and proper dietetic management. Under the best conditions, however, the cure will be slow and the physician and parents must show both patience and perseverance.

The *prognosis* is always grave, but it depends much upon the care the child can receive. Many of the cases that die annually could be saved if they could be removed to more favorable surroundings and receive more skillful and conscientious nursing. Persistent watching and self-sacrifice on the part of the mother or nurse will often apparently accomplish the impossible in these cases.

Diagnosis.—The differentiation between marasmus and *tuberculosis* may present difficulties. It is said that the tuberculous infant is bright in appearance and not so prostrated and apathetic as the marantic infant, but this is not a reliable sign. In tuberculosis we have continued fever as a more or less con-

stant symptom. Furthermore, repeated careful examinations of the chest will ultimately reveal evidence of tuberculosis and we may also be able to detect enlarged mesenteric glands by palpation of the abdomen. Persistent diarrhea with pus in the stools and at times blood speaks strongly for tuberculosis. A history of tuberculosis in the parents or of exposure to tuberculosis is suggestive of tuberculosis. Finally, a positive von Pirquet reaction is definite clinical evidence of an active tubercular infection at this time of life.

Hereditary syphilis must be excluded in all cases of malnutrition in infancy before a diagnosis of marasmus can be made. The characteristic symptoms and stigmata of this disease may be absent and wasting and anemia may be the only evidence of a serious constitutional disturbance. A Wassermann test should therefore be made in all doubtful cases.

Malnutrition is a much commoner condition than marasmus. It may be the result of premature or inherited feebleness of constitution, or follow after some acute illness, notably a gastrointestinal affection. Again, malnutrition is a prominent symptom in tuberculosis, syphilis and rickets.

Its most usual cause is improper feeding and unhygienic surroundings. As to the last named factors, they are just as likely to be encountered in well-to-do families as among the poorer classes, for here proprietary foods and close, overheated nurseries may be etiological factors. In older children anemia and malnutrition often date back to an attack of one of the infectious diseases or result from improper eating and hygiene (see Chronic Intestinal Indigestion). The diagnosis of simple malnutrition rests upon the exclusion of an organic disease or infection of which it might be only symptomatic.

Treatment.—The regular weekly weighing of the infant is an absolute necessity and the only accurate guide by which we can judge of the progress of the case. Whenever a weight disturbance occurs, the evening and morning temperature should be taken regularly, as this will indicate whether or not

we must resort to artificial heat or extra clothing; also whether the infant must be kept in bed or taken out in the fresh air. With a persistently sub-normal rectal temperature I have found it best to keep the infant in its crib well clothed and a hot water bag at the feet. Such infants should not be bathed but gently washed and then rubbed with warm olive oil. Very young infants who are too much exhausted by dressing and undressing can be wrapped in raw cotton.

Dietetic treatment.—If the infant be breast fed we must determine by examination of the milk whether it is sufficient in amount and of proper chemical composition. If the milk be at fault and appropriate treatment applied to the mother does not improve the same, we must try a wet nurse. If the milk is simply deficient in quantity, mixed feeding should be instituted.

As it is not always possible to obtain a wet nurse, we should bear in mind that in modifying the milk for a delicate or marantic infant it must be of a strength that would be suitable for a much younger infant than the one in question.

In the artificial feeding of a marantic infant a reduction of the fat in the food is of first importance. This applies especially to cases in which *constipation* is a prominent symptom. When the symptoms are extreme and of long standing the best mode of procedure is to put the infant on churned buttermilk or fat-free lactic acid milk for about a week after which time a change to Keller's Malt Soup may be advantageously made.

Cases in which *diarrhea* is present do best on Finkelstein's Eiweissmilch. After the intolerance to sugar has been overcome a carbohydrate, preferably Dextrin-maltose, may be added. This should be added in small amounts (one level tablespoonful to 24 hour quantity of food) and gradually increased to three level tablespoonfuls in 24 hours.

Another food which frequently gives excellent results in cases showing marked intolerance to the fat of ordinary milk modifications is the Czerny-Kleinschmidt butter-flour mixture.

This may be prepared as follows: Two tablespoonfuls of butter are boiled for five minutes over a slow fire to drive off the volatile fatty acids and then two tablespoonfuls of wheat flour are stirred into the butter and twenty ounces of water are added. This is allowed to cook slowly for five minutes, occasionally stirring the mixture, after which ten ounces of previously boiled whole milk are added. Cane sugar may gradually be added beginning with one teaspoonful and increasing to two level tablespoonfuls.

An important point to bear in mind in feeding a marantic infant is to give it relatively small quantities of food at long intervals (4 hours) until its food tolerance has been restored. Even though the above mentioned foods are well adapted to the delicate digestion of such a case, still they must be cautiously administered and overfeeding with the same is to be strictly avoided. A rapid gain in weight must not be anticipated or expected until the infant's assimilation has been decidedly and permanently improved. A grave mistake which is often made is to attempt to feed these infants according to their caloric requirements and give them from fifty to sixty calories per pound of body weight. The invariable result is to aggravate the dyspepsia and cause them to lose more weight. We should be satisfied if these infants maintain a stationary weight for a time or make only a slight gain until their digestion has improved and they are in a condition to be fed up.

Stimulation is at times called for. A few drops of brandy, well diluted, given during periods of great depression, has seemed helpful. Panopetone may also be tried.

On account of the anemia, freshly prepared beef juice (diluted) should be given in small quantities daily (one to two teaspoonfuls). Diarrhea may temporarily contraindicate its use. We know that even human milk contains insufficient iron to supply the requirements of the organism after a certain period, as has been pointed out by Bunge, and that the infant actually draws from the store of iron present in its tissues at

birth to sustain the hemoglobin percentage of the blood. Consequently anemia develops if milk is continued as the sole food beyond a certain time, and more markedly in subnormal than in normal infants. Orange-juice should also be given for obvious reasons. Occasionally small amounts of cod liver oil will be tolerated and assimilated when cream has to be entirely eliminated from the diet.

In looking over the list of remedies recommended in disorders of nutrition, the deep acting constitutional ones stand in the foreground. Much benefit is derived, however, from paying attention to the acute symptoms as they arise and prescribing such remedies as *nux vomica*, *podophyllum*, *ippecac*, etc., intercurrently.

The *calcareas* seem indicated in the majority of cases, especially *calc. phos.* which is best indicated in undersized, marantic infants who give a history of previous attacks of diarrhea and congenital debility. *Calc. carb.* is better indicated in cases resulting from fat intolerance in which there is constipation of large putty-like stools, prominent abdomen and sweating about the head. *Alumina* should also be thought of in cases with obstinate constipation. *Iodine* is strongly related to emaciation and glandular atrophy, and the *iodides* are often indicated, especially the *iodide of arsenic*, when there is great prostration, nervous irritability and restlessness; tendency to diarrhea; dropsical swelling of the face and extremities.

Sulphur presents many of the symptoms of marasmus, and it suits especially the cases with cutaneous eruptions; intertrigo; irritating stools and urine (exudative diathesis). *Mercurius* is of decided value in cases presenting symptoms suggestive of syphilis.

Lycopodium and *natrum muriaticum* are important in malnutrition and emaciation, and will be suggested by their characteristic symptoms. *Lycopodium* has a dark-colored urine which stains the diaper and which becomes strongly ammoniacal at times. The child is voracious, exceedingly irritable and there is much abdominal distention.

RICKETS; RACHITIS.

Rickets is a chronic nutritional disease of childhood in which there is a pathological standstill in the normal process of ossification, with resulting softening and deformity of the entire osseous system. Malnutrition and certain general disturbances are associated with the osseous changes.

Rickets belong to the "deficiency diseases" probably resulting from a deficiency or total absence in the child's diet of the fat-soluble vitamin (vitamin A) which is found in the fat of fresh raw milk. Prolonged heat destroys this vitamin for which reason rickets is frequently encountered in infants fed upon proprietary foods, condensed milk and boiled or sterilized milk. Rickets may develop in infants at the breast if the mother's milk is deficient in fat or if the nursing is prolonged beyond the normal period. It is readily induced in young growing animals by feeding them upon a diet deficient in the fat-soluble vitamin. Mellanby (*London Lancet* 1919) succeeded in producing rickets in young dogs through depriving them of fat-soluble vitamin A. Hess and Unger (*Jour. Amer. Med. Asso.*, 1920) opposed the theory of avitaminosis as a cause of rickets in the human being on the grounds of not being able to improve their cases of rickets by means of a diet rich in vitamin A. Recently, however, Park and Howland (*Johns Hopkins Hospital Bull.*; 1921) have given both radiographic and clinical evidence of the uniform and consistent improvement which occurs in rickets from the administration of cod liver oil. They state that the changes in the bones which are brought about through the administration of cod liver oil amount to a complete cure, providing that the diet is not too faulty. The presumption is that the curative effect of the cod liver oil depends upon the fat-soluble vitamins which it contains rather than upon the oil itself.

Hereditary predisposition to rickets no doubt exists. The majority of infants escape the development of rachitic manifesta-

tions because they have obtained, through the fetal circulation, sufficient vitamin to maintain normal nutrition until they are weaned and put upon a mixed diet. Infants who lack this protective inheritance begin to show signs of rickets after the sixth month unless the diet is an exceptionally favorable one, namely one rich in fresh butter fat.

Rickets is essentially a disease of infancy. The earliest symptoms usually manifest themselves at the sixth month. If the condition progresses the disease will be fully developed when the child is a year old. Rarely the symptoms develop during the second year and persist into the third year. The so-called *fetal rickets* is not rickets but is a term sometimes used erroneously to designate cases of achondroplasia and myatonia congenita.

Unhygienic surroundings, poor ventilation, and insufficient fresh air and sunshine are important factors acting as predisposing causes. Geographically, rickets is confined to the temperate zone and is common among the Italians and negroes in the tenement districts of the large cities. Lack of sunshine is unquestionably a potent contributing factor in conjunction with insufficient fresh air. The greater prevalence of rickets during the winter months, its less frequent occurrence in southern countries and the beneficial effect of sunlight in the treatment of the disease, bear out this theory.

Pathology.—The earliest pathological disturbance encountered in rickets begins in the periosteum. This accounts for the general sensitiveness of the body and the disinclination on the part of the child to use its extremities and its discomfort on being handled. In the bones we find an irregular growth and distribution of the osteogenetic cells in the centres of ossification together with absorption and irregular deposit of lime salts.

The chemical composition of the bones is much altered. Thus, in the shaft of the tibia there is normally 21 per cent water, in rickets 45 per cent. In the ribs the percentage of water may be raised from 44 per cent (normal) to 66 per cent.

The most important alteration, however, is the decrease in calcium phosphate. The ash (mineral constituents) may fall from 60 per cent, which is about the average in normal bone, to 30 per cent or even lower. Such a bone can be readily bent or cut.

The first demonstrable microscopical changes take place in the periosteum, being in the nature of an abnormal proliferation of its cells. In the medullary canal, a fibro-cellular hyperplasia takes place which invades and replaces the medullary substance. The same process may affect the epiphyseal portion of the bone or even the diaphysis, leading to thickening and structural changes.

At the extremity of the long bone, where the shaft and epiphysis are joined, growth is most active, for it is by the formation of new bone from the proliferating cartilage cells and their ultimate calcification that the bone increases in length. At this point rickets shows its most marked effect upon osteogenesis. The proliferating zone of cartilage cells is increased as are also the rows of cartilage-cells columns, which at the same time lose their regular arrangement. The zone of temporary calcification encroaches upon the upper layers of the cartilage and becomes interspersed with a net-work of blood vessels, islets of uncalcified cartilage and osteoid tissue.

In the medullary canal of the shaft, absorption of lime salts takes place, the canal becoming abnormally large and the marrow being replaced with fibro-cellular and vascular tissue. The outer layers of the bone become thickened through excessive proliferation of the periosteum and the production of osteoid tissue. In the flat bones, particularly in the occipital bone, absorption of osseous tissue in small areas results in the production of craniotabes.

These alterations in the structure of the bone explain its alteration in shape namely the thickening of the shaft and the clubbing of the extremities, and also account for the pliability and consequent deformities of the long bones.

With the arrest of the rachitic process, calcification of the

cartilage sets in and the bone may become abnormally hard. The hypertrophic tissue in the centres of ossification and along the epiphyseal lines is absorbed to a great extent so that the only permanent deformity which ultimately remains, is the distortion and bending of the bone that took place during its soft stage.

The soft structures of the body contain a normal amount of lime salts.

Pathological changes in other organs are not characteristic and constant. The liver may be enlarged. Splenic enlargement, due to simple hyperplasia, is not uncommon. Anemia may be pronounced. Catarrhal processes in the gastro-intestinal tract and in the lungs may be associated with rickets.

Symptoms.—The typical symptoms of rickets are the osseous changes and deformities. These are, however, late manifestations of the disease. An early diagnosis of rickets rests upon a knowledge of the early general disturbances which should always be looked for in artificially fed infants.

Rickets seldom develop before the sixth month, being practically a disease of the first dentition period. Its onset is usually associated with dyspeptic symptoms, moderate range of fever, fretfulness, restlessness with tendency to kick off the covers, and excessive sweating. Sweating is most pronounced on the head but the body is often covered with sudamina and a miliary rash. The development of anemia, debility, profuse sweats and general sensitiveness of the body are indications of the onset of rickets. Constipation with abdominal distention is a characteristic late manifestation due to intestinal atony. Rolling the head from side to side until the hair has been rubbed from the scalp in the occipital region is another characteristic symptom. The hair is abnormally dry and brittle and is thus readily shed.

The entire muscular system is in an enfeebled undernourished condition. This accounts for the constipation, weak heart with sluggish circulation, and the rachitic pseudo-paralysis.

This latter condition results from the ligamentous laxity, muscular feebleness and the soft condition of the bones.

The first bone changes are usually found in the ribs. As the disease progresses the epiphyses of the long bones become involved. There is, however, no fast rule as to sequence in the development of the deformities, and it is rare to find all of the characteristic lesions in an individual case. The ribs become beaded in their anterior extremity, at the junction of the rib with the costal cartilage. This deformity is described as the *rachitic rosary*, and it can be demonstrated in almost every case on post-mortem dissection as well as recognized by palpation during life. Sometimes the rosary can be plainly seen. Owing to the softening of the ribs, the thorax becomes compressed laterally, with resulting projection of the sternum; this is the *pectus carinatum*, or chicken-breast. Another deformity of the chest is a groove encircling the lower portion of the thorax, the so-called Harrison's groove. This line corresponds with the lower border of the lungs and it is produced by recession of the lateral region of the soft, yielding thorax from atmospheric pressure, and the eversion of its lower border due to the large, distended abdomen. These deformities become especially prominent as a result of diseases of the respiratory tract.

Affections of the cranial bones are among the earliest signs of rickets. Softening of the occiput, with areas of craniotabes, can be demonstrated, especially in the region of the lambdoital suture. The occipital region becomes flattened as a result of the child lying on its back. The sutures are late in closing, the fontanel abnormally large, and the frontal and parietal centres of ossification are prominent. These developmental peculiarities give to the head a large, square appearance, very typical of rickets. The head may also become misshapen and asymmetrical from lying more on one side than upon the other during the stage when the bones are soft and yielding.

The softness of the bones of the palate and of the jaw pre-

disposes to the development of deformities from the act of sucking and mastication.

The spinal column suffers more or less in all cases of rickets. Owing to the softness of the vertebræ and weakness of the spinal muscles and lax ligaments, the child develops a kyphosis, when sitting up, which may result in a permanent deformity if the condition is not recognized and corrected. Rachitic kyphosis presents a curved outline, involving the greater portion of the spinal column, and in its early stages it can be entirely reduced by laying the child upon its stomach and making traction on the spine by lifting it up by the legs. The deformity of Pott's disease is permanent, angular in outline and involves only one or two vertebræ. Scoliosis may also develop in the rachitic infant.

The extremities suffer from bending and twisting, as a result of muscular traction and the weight of the body. The humerus and tibia suffer most frequently. Serious deformity of the pelvis rendering parturition difficult or even impossible is one of the unfortunate late results of rickets.

The eruption of the teeth is delayed and irregular, and they may decay early on account of a deficiency of or irregular deposit of enamel. Rachitic teeth are typically ridged in their axis and sometimes present a saw edge. They must not be mistaken for syphilitic teeth.

Rachitic children show a marked predisposition to a variety of ailments, referable to the nervous system, the alimentary tract, the skin and mucous membrane. They are also subject to catarrhal disturbances and to bronchitis.

Among the disturbances of the alimentary tract complicating rickets, chronic indigestion, chronic intestinal catarrh and obstipation are of the most common occurrence.

The nervous system is particularly unbalanced and highly susceptible to peripheral impressions. Trifling ailments are liable to be ushered in with convulsions, and, in fact, convulsions occurring after the first year should always lead to a

suspicion of rickets. Spasm of the glottis and tetany occur more frequently in rachitic infants than in non-rachitic. There is a close relationship between spasmophilia and rickets owing to the fact that both present as an underlying condition a serious disturbance of calcium metabolism.

The alterations in the blood are not constant and uniform. In all cases more or less anemia is present, and in some, especially those with splenic tumor, there may be leucocytosis with abnormal elements (myelocytes; mast-cells and normoblasts) in the blood. The hemoglobin and red cells are diminished in varying proportions.

The course of rickets is chronic but the early institution of treatment, together with the favorable influence of fresh air and sunshine, will, as a rule, bring about prompt amelioration of the symptoms.

The *differential diagnosis* lies between *hereditary syphilis*; *hydrocephalus*; *Barlow's disease* or *infantile scurvy*; and *Pott's disease*. The differentiation from the last condition has already been considered above. *Barlow's disease* is a more acute disease, more frequently found in infants of the better classes as a result of exclusive feeding with proprietary foods, and is attended by swelling of the shafts of the bones from subperiosteal hemorrhages; joint tenderness and swelling and ecchymoses in various parts of the body and hematuria. Scurvy is frequently associated with rickets and it should be suspected when the rachitic symptoms become acute in character.

Epiphyseal disease and separation is a symptom of *congenital syphilis*, which, however, occurs in the earliest months of life, other signs of syphilis being demonstrable.

Chronic hydrocephalus presents a head more rounded than the rachitic cranium; the face is disproportionately small in comparison with the head; the eyeballs are deflected downwards, and the mental condition is one of dullness and imbecility rather than precocity, as in rickets.

The *prognosis* is favorable if the disease is recognized

early and if proper treatment can be carried out. An uncomplicated case without pronounced deformity of the chest, anemia and splenic tumor is generally promptly amenable to treatment, while those with such unfavorable symptoms especially in conjunction with laryngismus stridulus present a more serious prognosis. The occurrence of pneumonia or whooping-cough in a rachitic infant is a grave complication.

Treatment.—Prophylactic measures should be instituted during pregnancy if a hereditary predisposition to rickets has been noted. The mother should take a diet containing sufficient milk, cream and green vegetables to supply the fetus with the necessary vitamins. The use of condensed milk and proprietary foods in the child's diet, in the place of modified fresh cow's milk, should be discouraged. The early addition of fruit juices and vegetable broths to the infant's diet should be prescribed. Ample fresh air and sunshine are absolutely essential to the infant's welfare.

Cod liver oil, owing to its vitamin content, is an excellent addition to the diet and may be looked upon as a specific for rickets. The raw yolk of egg may also be used with advantage.

In the early stages of rickets *calc. phos.* is the chief remedy. The scrawny, under-developed infant, with flabby abdomen; diarrheal stool containing greenish mucus, undigested casein and fat particles; delayed teething and large fontanel; closely corresponding to the incipient period of the disease. Later on, as the osseous changes, the anemia, local sweating about the head and the distended abdomen become prominent symptoms, *calc. carb.* is more applicable.

Kassowitz demonstrated that *phosphorus* exerts a specific, selective action upon the epiphyses of the long bones, inducing an inflammatory process of the bone-forming cartilage at this point thus presenting a strong similarity to the rachitic process. On the strength of this he was the originator of the "phosphor-therapie" in rickets, being championed by such pediatricists as Demme, Soltmann, Jacobi and others. *Phosphorus* may, there-

fore, be confidently used as a homeopathic remedy for rickets. Babinsky has observed that it is most helpful in cases with laryngismus stridulus.

Ferrum phos. is a valuable remedy for the acute respiratory disturbances of rickets. It is also useful in the anemia of rickets and may be used in conjunction with *calcareo phos.* in the early stages of the disease.

The following remedies should be studied for special indications:

Alumina.—Abnormal cravings or voracious appetite; open fontanels; distended abdomen; obstipation, from inactivity of the rectum.

Bell.—The nervous manifestations of rickets frequently call for this drug.

Kali hydrojod.—Preliminary symptoms of rickets. Tenderness of the entire body, but especially about the head (Cooper).

Mercurius.—Cases with a syphilitic family history.

Silica.—Profuse sweating about the head and chest, with general sensitiveness of the body; anemia; pale skin through which the bluish veins are prominently seen; swelling of the epiphyses of the bones and affections of the cartilages in general; skin dry and scaly, with tendency to suppurative affections, notably paronychia; imperfect assimilation; child is slow in walking.

INFANTILE SCURVY; BARLOW'S DISEASE.

Infantile scurvy, also known in the literature as Barlow's disease, is a chronic nutritional disturbance belonging to the deficiency diseases which develop as the result of a diet deficient in antiscorbutic vitamin C. It was long ago suspected that the feeding of sterilized milk, condensed milk and preserved proprietary foods was responsible for the majority of cases of scurvy. Even recent investigations into this subject trace the immediate cause to the use of pasteurized milk or foods prepared with boiled milk (Hess and Fish, *Amer. Jour. Dis. Child.*, Dec.,

1914). The two-fold heating of milk, that is, boiling or re-pasteurizing a pasteurized milk, is an important etiological factor. The early symptoms have been frequently controlled by simply substituting raw milk for pasteurized milk. The exclusive use of patent foods is a notorious cause for scurvy, as was shown by the American Pediatrics Society's investigation in 1898.

Notwithstanding the undeniable relationship between scurvy and milk that has been subjected to a high temperature, nevertheless, other factors appear necessary in the etiology of the disease for by no means all infants thus fed become scorbutic. I have seen several cases of scurvy develop in infants who were fed with raw milk, but the cows from whom this milk came probably did not receive any fresh, green fodder. On the other hand, in some communities it has long been the custom to feed infants with boiled milk and yet scurvy rarely develops there. This applies especially to mild and warm climates, where, incidentally, rickets is also less common than in the colder climates. Perhaps the early resort to mixt feeding in these countries and the transmission of vitamins through the placental blood in sufficient amount to ward off scurvy explains these apparent discrepancies. No doubt the mother's diet during pregnancy has some influence on the etiology. According to the observations of Hess, there is a strong relationship between the exudative diathesis and scurvy, although there is no direct relationship to rickets.

Age is an important predisposing factor. The majority of cases are encountered during the second half of the first year. It is rare before the sixth month or after the eighteenth month.

Symptoms.—The characteristic symptoms of scurvy in infancy are anemia; sponginess and bleeding of the gums; subperiosteal hemorrhages notably of the lower extremities; general sensitiveness of the body, and pseudo-paralysis of the extremities.

The early manifestations of scurvy are a moderate amount

of fever and painfulness of the extremities, most marked about the epiphysis of the bones. The child often shows the early symptoms of rickets, as an associated manifestation of faulty hygiene and feeding. A failure to gain weight and a standstill in the child's growth as a rule precede the appearance of the hemorrhagic manifestations. Anorexia is a frequent early symptom and when this is noted in a child that is fretful, pale, not gaining in weight and loath to be handled, we have a symptom complex which is strongly suspicious of beginning scurvy.

Associated with the hypersensitiveness of the extremities we may soon detect a swollen, purplish appearance of the gums and perhaps small petechial spots on the body. As the disease progresses a distinct swelling can be made out in one of the extremities. The most frequent site for the swellings, which are due to sub-periosteal hemorrhage, is the lower extremity of the femur. Both legs may be involved. The frequent handling of the legs in changing the diaper may be responsible for the hemorrhage. Another favorable site for the sub-periosteal hemorrhage is the lower end of the tibia. The epiphysis may be involved to a degree resulting in epiphyseal separation. Epiphyseal hemorrhage is an early symptom and may be recognized by the X-ray as the "white line" of Frankel.

Hematuria is at times the first symptom observed, and together with tenderness of the body may be the only symptom present. Morse has reported several such cases, and Barlow himself recognized this fact at the time he brought the disease before the notice of the profession.

The course of scurvy is an acute one, and under proper treatment it can be shortened to a few weeks. Fatal cases have occurred, especially in those whose true nature was not recognized in time. While the acute manifestations of the disease promptly subside upon the institution of antiscorbutic treatment, still it may require weeks and even months before a large, organized sub-periosteal clot is completely absorbed.

The *diagnosis* is not difficult when the characteristic symptoms have developed. Hematuria in infancy should always be looked upon as strong presumptive evidence of scurvy, and the application of the therapeutic test, namely, fresh milk and orange juice, should be employed in such cases.

Syphilitic epiphysitis occurs in younger infants, usually from two to four months old; it involves, by preference, the lower end of the radius or is multiple and symmetrical and is associated with snuffles and skin and mucous membrane lesions.

Articular rheumatism is more frequently diagnosed in cases of scurvy than any other condition. Such a diagnosis indicates carelessness on the part of the physician, for there is no arthritis in scurvy but a lesion of the shaft of the bone at the epiphysis and above the same. Furthermore, articular rheumatism is practically unknown in infancy. An arthritis in infancy is, as a rule, septic or gonococcic in origin.

Treatment.—In the treatment of scurvy a change of food is demanded at once. A milk formula suitable to the child's age, preferably unsterilized, and fed in definite quantity and at regular intervals, together with the administration of fruit-juice (three to four teaspoonfuls of orange-juice twice daily), are the prime dietetic requirements. Other valuable additions to the diet are potato, tomato juice and green vegetables. In the case of young infants a small amount of baked potato may be mixed with the milk while in older infants it may be given as a vegetable. Fresh meat-juice should also be given at regular intervals if anemia and prostration are marked.

Constitutional remedies are of value, particularly for the malnutrition. The child's suffering can also be alleviated by remedies covering the acute symptoms such as *ferrum phos.*, *bryonia*, *rhus tox.*, *ruta* and *mercurius*.

As a constitutional remedy *phosphorus* should be prescribed. This remedy covers the pathological changes encountered both in the blood and in the osseous system.

EXUDATIVE DIATHESIS.

The term exudative diathesis was coined by Czerny to indicate an abnormal type of constitution in consequence of which the infant becomes susceptible to various skin and mucous membrane affections characterized by exudation and inflammatory reactions. The skin presents a predisposition to scaling, erythema, papular lesions (lichen strophulus) and eczema. The scalp is affected with seborrhea while on the face a moist type of eczema develops. Wherever the skin is thrown into folds, about the neck and buttocks especially, a severe type of intertrigo develops.

The mucous membranes are prone to catarrhal inflammation and rhinitis and recurring bronchitis are common symptoms. Lingua geographica is observed at times. Dyspeptic disturbances with mucus in the stools is a common occurrence. There is also a tendency to swelling of the superficial lymph nodes while the tonsils and pharyngeal adenoid tissue are usually hyperplastic.

Infants presenting symptoms of exudative diathesis are usually plump and well nourished in appearance suggesting a case of overfeeding. They are susceptible to infections and frequently a moderate degree of irregular continued fever is noted, suggesting a tubercular infection. The temperature, however, is due to a dyspeptic condition or a naso-pharyngeal affection and a negative von Pirquet reaction as well as the subsequent course of the case serve to exclude tuberculosis. Occasionally an infant showing the clinical manifestations of the exudative diathesis will be thin and undernourished but as a rule they are fat.

The blood shows an eosinophilia which is the most characteristic and suggestive clinical manifestation of the condition. This would indicate that the child is suffering from a chronic protein sensitization resulting from an intolerance to cow's milk or to overfeeding with the same. The fact that an infant

with symptoms of the exudative diathesis promptly improves when the diet is changed from milk to cereals and vegetables tends to confirm the belief that it is a condition of intolerance to the protein of cow's milk. Similar symptoms may develop in a breast-fed infant as a result of overfeeding plus infection, the anaphylaxis being of bacterial origin as in some cases of bronchial asthma or the sensitization may occur through the breast milk from certain foods in the mother's diet (O'Keefe).

The first step in the *treatment* of manifestations of the exudative diathesis is to cut down the amount of food, reducing the feedings to four to five daily and substituting cereals, vegetable soup and strained vegetables for one or two of the milk feedings. It is also well to skim the milk; two to three teaspoonfuls of cod liver oil each day can be substituted for the cream. Buttermilk in place of sweet milk may also be given at times with good results.

SPASMOPHILIA; TETANY.

Spasmophilia, or the spasmophilic diathesis is a condition of abnormal mechanical and electrical irritability of the peripheral nerves associated with a tendency to localized and general convulsions. It is frequently encountered in infancy both in a latent and active form and there is a distinct seasonal occurrence during the colder months of the year. It is practically never encountered in breast-fed infants. Etiologically it is a disturbance of calcium metabolism resulting from a dietetic error or from inability of the infant to adjust itself to cow's milk together with a deficiency of fresh air and sunshine as its seasonal incidence suggests. Evidences of rickets are frequently associated with spasmophilia.

The majority of convulsions occurring in infants are due to spasmophilia. Attacks of spasm of the glottis, rotary head spasm and tetany are also manifestations of spasmophilia. Striated muscles alone are affected; the spasmodic symptoms encountered in the "hypertonic infant" which involve the

gastrointestinal tract to a large extent producing pylorospasm and intestinal colic are manifestations of vagotonia and not of spasmophilia.

The most constant symptom of spasmophilia is the heightened electrical irritability of certain motor nerves (Erb's phenomenon). This is elicited by placing one electrode over the chest or abdomen and the other over the median nerve in the bend of the elbow. The peroneus nerve may also be used in making this test. In a normal infant the cathodal opening contraction is brought about with a current of 5 milliamperes while in spasmophilia 3 to 4 milliamperes will produce a sharp contraction.

The mechanical irritability of the peripheral nerves is demonstrated by striking the nerve or one of its branches a quick blow with a percussion hammer or with the finger; contraction of the muscles supplied by the nerve results from this stimulus. The most characteristic response is seen when the facial nerve is irritated by a light blow on the cheek, this being followed by a quick contraction of the face muscles (Chvostek's sign).

Another diagnostic sign of spasmophilia is Trousseau's phenomenon which is the production of a tonic spasm of the muscles of the hand and forearm in the position seen in tetany by compression of the nerves and bloodvessels in the bicipital groove. There is some danger, however, of bringing on an attack of laryngospasm in eliciting this phenomenon and so caution should be exercised in making use of this diagnostic sign.

The chief clinical manifestations are general convulsions, spasm of the glottis and tetany.

General convulsions, or *infantile eclampsia* are attacks of epileptiform convulsions with clonic spasms of the entire skeletal muscular system associated with loss of consciousness. The convulsive manifestations usually last for several minutes and there is a strong tendency for the same to recur. The age of the patient, the tendency to repetition of the attacks and the

presence of the stigmata of spasmophilia serve to differentiate infantile eclampsia from epilepsy. The convulsions are usually precipitated by some source of local irritation such as a gastrointestinal upset or teething; the onset of an acute infection is also liable to be ushered in with a convulsion.

Often, however, the convulsions cannot be assigned to any demonstrable cause. They may recur at frequent intervals or occur only once as the result of an acute digestive disturbance or fever. While convulsions are always alarming and cause the parents the greatest concern, still they rarely prove fatal in themselves and are not as dangerous to life as spasm of the glottis. When convulsions continue uninterruptedly, however, assuming the status epilepticus the condition becomes grave. Death from uncomplicated convulsions is rare. A child may apparently die from a convulsion but an autopsy will demonstrate the status lymphaticus, meningitis, or a severe infection (streptococcic or pneumococcic).

Spasm of the glottis, or *laryngospasm* is mainly observed in rachitic infants; it is rarely observed in children over two years old. The symptoms of laryngospasm are a sudden cessation of respiration accompanied by ineffectual efforts to inspire and symptoms of asphyxia. The child may lose consciousness and become limp and near death. At this stage it may die or the spasm relaxes, inspiration accompanied by a crowing sound occurs and consciousness returns. In fatal cases death is usually attributed to cardiac paralysis rather than to asphyxia (heart tetany). It is a question, however, whether the heart is directly affected by the spasmophilic condition. The most plausible explanation attributes the infant's death in these cases to an expiratory apnea. Mild forms of laryngospasm are also encountered which manifest themselves as a crowing respiration noted whenever the child becomes excited or cries. While this condition is of no importance in itself, still notice should be taken thereof as it may be the forerunner of more serious symptoms.

Tetany, or *carpopedal spasm* is the most striking manifestation of the spasmophilic diathesis and presents a unique clinical picture. There is a persistent contraction of the flexor muscles of the hands and feet fixing them in a characteristic position. The hands are flexed at the wrist, the fingers are extended and flexed at the metacarpal-phalangeal joint and held in firm apposition to the thumb, giving the hand the so-called "obstetrical position." The feet are extended and held as in talipes equinus with the toes flexed like the fingers. The spasm comes on suddenly involving first the fingers and wrists, after which the ankle-joints and toes become fixed. Older children may complain of numbness and tingling in the extremities preceding the attack. A cramp-like pain in the muscles may be complained of and attempts to overcome the contracture are evidently painful. The spasm may extend to other groups of muscles so that the extremities may become involved and opisthotonus develop. Strabismus is noted at times. When the facial muscles are involved the features assume a peculiar, fixed expression. The attack may only last a few hours or it may persist for several days at a time. Frequently the attacks are intermitting.

Tetany is differentiated from tetanus by the absence of trismus, which is usually the first symptom of tetanus. It may be confused with cerebral diplegia or Little's disease but these are chronic affections of long standing with characteristic signs of upper neuron involvement and they do not present the characteristic carpopedal type of contracture seen in tetany or Chvostek's or Trousseau's signs.

The prognosis for the ultimate disappearance of the manifestations of spasmophilia is good if the child can receive the proper hygienic and dietetic treatment. Of first importance is abundance of fresh air and sunshine. There is a spontaneous improvement in all cases during the summer months undoubtedly due to the outdoor life and sunshine which the infant naturally gets at this time of year.

In regard to diet, breast-milk gives the best results and may be looked upon as a specific. However, the difficulty of obtaining a wet nurse usually puts this method of feeding out of the question. Cow's milk may be continued in the case of young infants the daily quantity being limited to 16 to 20 ounces and part of the cream removed. Cereals and vegetables (strained vegetable soup) must be added to the diet as soon as possible, substituting a feeding with cereal or soup for one of the customary bottles. If the infant is constipated a malt soup formula should be used in place of a simple milk modification. Cod liver oil and *phosphorus* are the best therapeutic agents. Three to four teaspoonfuls of cod liver oil with a little orange juice may be given daily in conjunction with a drop of the homeopathic tincture of *phosphorus* three times daily.

STATUS LYMPHATICUS AND ENLARGEMENT OF THE THYMUS GLAND.

Enlargement of the thymus gland in infancy is looked upon as a clinical manifestation of the status lymphaticus although the condition is frequently encountered in the absence of any symptoms of the lymphatic diathesis. On the other hand, the typical case of the so-called status lymphaticus is one which has never shown symptoms of an enlarged thymus. Such an infant, in the midst of apparent health and with a negative past history, may suddenly develop a high temperature accompanied by convulsions and die within less than twenty-four hours. Another evidence of this diathesis is sudden death during an anesthetic or even sudden death without any discernible cause. The autopsy findings under these circumstances may confirm the suspicion of the status lymphaticus as the determining cause of death. The pathological changes encountered are a universal hyperplasia of the lymphoid tissue throughout the body most strikingly seen in the enlargement of the bronchial glands, the mesenteric glands and the solitary follicles of the small intestines. There is enlargement of the spleen and hyper-

trophy of the tonsils. Sometimes lymphoid tissue is found in the various organs such as the liver and kidneys. The thymus gland is almost always found enlarged in these cases although there may have been no symptoms referable to the thymus during life.

The thymus gland at birth is slightly larger than in later infancy and it undergoes a gradual involution so that at five years it weighs 4 grams as compared to 6.5 gms. at birth. According to Bovaird and Nicoll a thymus of 10 gms. and over may be considered abnormally large. Cases have been recorded in which the gland weighed from 30 to 40 gms.. In such instances the general lesions of status lymphaticus, namely, marked hyperplasia of the tracheobronchial lymphnodes and a general hyperplasia of the lymphoid structures throughout the body are usually encountered. An enlarged thymus can usually be palpated in the suprasternal notch. Prominence of the sternum may also be noted.

Percussion of the normal thymus is not possible. Dulness in the region of the gland is evidence of hypertrophy of the same. The dimensions of the normal thymus are two to three centimetres in breadth and about five centimetres in length. The area of dulness over an enlarged thymus exceeds these dimensions considerably.

The *symptoms* directly due to the enlarged thymus are dyspnea and a laryngeal stridor occurring either intermittently or more or less constantly present. In the milder cases the dyspnea is only noted when the child cries or becomes excited; in the more severe cases a constant difficulty in breathing is noted. The respiratory embarrassment may be so pronounced as to result in attacks of cyanosis. The dyspnea is inspiratory in character and is aggravated by moving the head backward.

Another prominent symptom is a persistent cough occurring in a young infant without any demonstrable cause. It is dry and paroxysmal in character and may have been noted from birth. Sibilant rales may be heard in the chest, probably

resulting from pressure upon a bronchus or from irritation of the broncho-constrictor fibres of the vagus. In one of my cases the child died in one of its suffocative attacks and at autopsy the trachea was found flattened out by the pressure of an unusually large thymus.

As a rule the symptoms of respiratory embarrassment and the cough are present from birth but at times these symptoms do not develop until a later period. A cold or an attack of bronchitis may first call attention to the condition and the persistence of the symptoms will arouse the suspicion of an enlarged thymus.

The *diagnosis* of enlargement of the thymus gland in well marked cases should not be difficult. We should suspect the condition when the characteristic symptoms above enumerated are encountered, namely:

1. Recurring attacks of dyspnea which cannot be attributed to adenoids or other nasopharyngeal obstructions, e.g. retropharyngeal abscess, the dyspnea being inspiratory and aggravated by hyperextension of the head.

2. Persistent cough and stridor in a young infant, present since birth or developing later in infancy, with inspiratory dyspnea and without fever. The presence of fever and expiratory dyspnea suggest bronchial gland tuberculosis.

The diagnosis can usually be verified by the following signs:

1. Dulness over the upper portion of the sternum.
2. Resistance in the suprasternal notch, increased during the expiratory phase.
3. X-ray demonstration of an increased shadow to either side of the sternum, overlying the great vessels.

Treatment.—The results obtained by the X-ray treatment of enlarged thymus gland are most encouraging. I have seen prompt and lasting improvement in the pressure symptoms in a number of cases treated in this manner. There is no doubt in my mind that the X-ray causes a decrease in the size of the

gland judging from the relief of the symptoms following its employment. The technique is as follows:

The child is treated every three weeks, three minute exposures being given both front and back with a Coolidge tube of $8\frac{1}{2}$ inch vacuum, used at a distance of 8 inches, the rays being filtered through 3 millimeters of aluminum (Dr. J. W. Frank). Often improvement will be noted after the first treatment.

Surgical interference may become necessary when the pressure symptoms become alarming. Intubation or tracheotomy are valueless because the tube does not reach far enough down into the trachea. Partial resection of the gland, or anchoring the gland to the upper portion of the sternum has given relief.

Constitutional treatment should be carried out in conjunction with the X-ray or surgical treatment. The associated bronchitis usually requires attention and *belladonna* will relieve the cough and wheezing. The *lime salts* are valuable especially since the thymus is so prominently associated with calcium metabolism. A 3x trituration of the *iodide of lime* is the preferable form in which to use this salt. Cod liver oil should be given in conjunction with the *calcareo iodide* because of its beneficial effect on calcium metabolism.

TUBERCULOSIS.

Tuberculosis in infancy and childhood is usually encountered in the primary stage of the infection. In infants there is a tendency to a rapid spread of the process to contiguous structures owing to the infant's lack of resistance to tuberculosis and therefore the evidences of a secondary infection are soon demonstrable. In older children the infection is, however, usually held in check by the regional lymph-nodes and a secondary invasion of the lungs or a general infection is thereby prevented. For this reason tuberculosis in infancy presents a high mortality rate because of the infant's lack of resistance to the infection

while in childhood the mortality is low and the disease tends to become localized and remain latent.

On the basis of these observations it is generally held, in reference to the etiology of pulmonary tuberculosis, or consumption, that the seed for the same is sown in early childhood and that the pathological changes which occur in the lungs in a case of consumption do not correspond to the pathology of a primary lung infection but represent the reaction of a partially immunized individual to subsequent infections.

There is probably no natural immunity against tuberculosis. Infants appear to be particularly susceptible to the tubercle bacillus and evidently exposure to infection is all that is required in order to contract the disease. Arrested infections, however, acquired during early childhood, appear to confer more or less immunity against subsequent infections in later life. Such an infection at least tends to develop resistance against the spread of the disease throughout the body and thereby lessens the chances for the development of acute miliary tuberculosis or tuberculous bronchopneumonia. This assumption is based upon the fact that tuberculosis is usually of the disseminated type in infancy while during later childhood it tends to become localized. In this localized, or glandular form it may become dormant and eventually undergo spontaneous cure. If, however, a child with such a latent infection is re-infected or its vitality is depressed by an intercurrent disease or through improper food and unhygienic surroundings, a new tubercular process, modified by the partial systemic immunity toward the tubercle bacillus, develops. Such a process does not present the manifestations of an acute miliary or caseous tuberculosis but it is characterized by a chronic course and shows attempts, more or less successful, on the part of the host to overcome the infection (see "Tuberculosis of the Lungs").

Childhood furnishes the majority of cases of localized tuberculosis, namely, tuberculosis of the glands, bones and joints.

The scrofulous child is one that has a latent infection, usually glandular, which more or less protects it against a general infection, but which has made it hypersensitive to the toxin of the tubercle bacillus. These children develop severe reactions on the mucous membranes and skin and give a strong von Pirquet reaction. They are also liable to phlyctenular keratitis. The majority of children over two years of age who develop tuberculous meningitis and acute general tuberculosis show no evidence of previous infection. They do not give the von Pirquet reaction and at the autopsy there is rarely evidence of an old lesion. In children under two years of age however tuberculous meningitis and acute miliary tuberculosis occur as the so-called second stage of tuberculosis and develop as the result of a primary lung focus which the young organism is unable to hold in check. Infants appear to be unable to develop any degree of immunity against tuberculosis such as older children acquire from a small focus. The lymph nodes may hold up the infection for a while but as a rule it soon passes this barrier and extends into the pulmonary structure or gains entrance into the circulation setting up a generalized infection.

The percentage of *latent tuberculosis* among children of apparently normal health is strikingly high. Before the discovery of the cutaneous tuberculin test we could only surmise but not prove that a child harbored the tubercle bacillus somewhere in its body. This was the only explanation for the sudden development of active tuberculosis in a child previously apparently healthy after an attack of measles or whooping-cough or a rapid breakdown during adolescence incident to unhygienic surroundings, overwork at school, or in factories, insufficient food, etc.

In 1907 von Pirquet announced his cutaneous tuberculin test to the profession as a safe and practical method of demonstrating the presence of tuberculous infection in the individual reacting to this test. The fact that the reaction may be present even some time after the lesion has healed, owing to the pres-

ence of immune bodies in the tissues, makes the test of questionable value in adults but does not lessen its usefulness in childhood.

Von Pirquet found that in a series of 1,134 children in Vienna, clinically non-tuberculous, the reaction was found in percentages which rapidly rose, almost with step-like regularity, from 15 per cent at two years to 90 per cent at fourteen years. Mortality figures in tuberculosis strangely contrast with these findings. Death from tuberculosis in childhood is highest in early infancy. This is due to the infant's close proximity to the parent or nurse who may have tuberculosis and its close confinement to the house. Also to the fact that infection at this stage tends rapidly to become general.

The mortality falls decidedly after the third year and the lowest figures are reached between the fifth and tenth years. It does not materially rise again until the time of puberty at which epoch phthisis becomes a common disease.

How does the child become tuberculous? All signs point to infection by the way of the respiratory route, the source being an individual with open tuberculosis. Other forms of infection occur but they are rare. Of these, infection through the alimentary tract is of first importance. Primary intestinal tuberculosis is far less common than primary pulmonary infection, the chief reason, perhaps, being that it requires an enormously larger number of bacilli to set up an infection when the bacilli enter by the way of the alimentary tract than when they are inhaled, this has been proven by animal experiments. No doubt the same condition holds good in the case of human beings. The milk supply evidently plays an important role in the etiology of intestinal tuberculosis and the bovine type of bacillus has been identified in such cases.

Infection may take place through the mouth and tonsils and this explains the mode of occurrence of cervical and sub-maxillary tuberculous adenitis. In cases showing involvement of the supra-clavicular glands there is an associated apical pleurisy.

The bronchial glands, however, are the site of the infection in the great majority of cases irrespective of whether the child is clinically tuberculous or not. Most pathologists are of the opinion that the changes in the bronchial glands are secondary to a focus in the lung. This could not well be otherwise judging from the course pursued by an infection with the tubercle bacillus in other regions of the body.

Since the bronchial glands show the most advanced changes in the majority of autopsies, a belief in the preponderance of respiratory infection is justified. When the chain of glands on the right side is involved a primary focus can be found in the right lung and vice versa. Infection of both chains can only occur when there is a primary lesion in both lungs.

Bronchial gland tuberculosis is, therefore, the most important clinical variety of tuberculosis in childhood. If such an infection is latent it can only be suspected from the presence of a positive von Pirquet reaction. Enlarged bronchial glands may, however, frequently be demonstrated by percussion and auscultation, according to the method of d'Espine.

The sign to which d'Espine called attention is dulness between the shoulder blades, most marked from the 2d to the 4th dorsal vertebræ, associated with exaggerated transmissions of the whispered voice over this area. If the child is made to speak in a low voice an accompanying whispering sound is heard. The respirations are also distinctly bronchial over this area. The bifurcation of the trachea is on a line with the 3d dorsal spine and here the main bronchial glands are situated.

Enlarged bronchial glands may also produce obvious symptoms, the recognition of which makes the diagnosis possible independent of physical diagnosis and the cutaneous reaction. The symptoms referred to are a high pitched metallic cough, associated with expiratory dyspnea. This syndrome is seen typically only in infancy, at which age the presence of tuberculides is also a frequent aid in the diagnosis of tuberculosis. Tuberculides are small, hard papules, about the size of a pin

head, bearing a central depression. They are scant and may be found upon the trunk and extremities. Their clinical importance was first pointed out by Hamburger, one of Pirquet's associates.

Owing to the great prevalence of latent tuberculosis in childhood we should always bear it in mind in the presence of obscure fevers and in all cases of malnutrition and anemia. The mistake, however, is often made of ascribing to tuberculosis recurring or continued fevers due to a focal infection, empyema, rheumatism and intestinal toxemia.

The *symptoms* of tuberculosis in childhood depend upon the stage of the infection and the severity of the same. In the primary stage there is usually a continued irregular fever, loss of weight and anemia. These symptoms are more marked in infants than in older children. At this age the infection tends to spread rapidly to adjacent tissues and *tuberculous bronchopneumonia* or *caseous pneumonia* frequently develop within a short time after the bronchial glands have been infected. In older children, however, the primary infection is usually held in check by the lymph-nodes. There is, therefore, less danger of a general infection and the child may show no characteristic clinical manifestations which would suggest tuberculosis. A child with infected lymph-nodes, however, is likely to present a slight evening rise of temperature and show some evidence of malnutrition and anemia. A positive von Pirquet reaction and the d'Espine sign can usually be demonstrated. The tuberculous child is characteristically of slight build and is mentally alert; the skin is of fine texture and transparent and there is a tendency to excessive growth of hair between the shoulder blades and over the shoulders. There is usually a positive tubercular family history in such cases and an evident hereditary predisposition to pulmonary tuberculosis. Another physical feature of these cases which no doubt predisposes them to phthisis is the long, narrow, flat chest and the abdominal ptosis.

Acute miliary tuberculosis develops most frequently in infants as a result of general infection from a primary focus in the lung and bronchial glands. It is characterized by a high, continued fever of irregular type with rapid emaciation and the predominance of either respiratory or nervous symptoms according to whether the lungs or meninges are chiefly involved.

Scrofula is a chronic form of tuberculosis in which there is a tendency for the process to remain localized in the lymphatic glands or bones. The scrofulous child is coarse featured, not as bright mentally as the tuberculous type and it is predisposed to chronic skin and catarrhal affections (Exudative Diathesis). The prognosis as to life is much better in this type of tuberculosis which usually affects the lymphatic glands, particularly the cervical group, than in the type with a primary pulmonary infection. It is true, the bronchial glands arrest the infection in many instances but there is always the danger of this defensive barrier breaking down or of subsequent infections occurring with the consequent development of the chronic form of pulmonary tuberculosis.

Tuberculous Adenitis.—Tuberculous adenitis is distinguished from simple adenitis by its chronic course and clinically by its predilection for involving the anterior cervical lymph nodes. It is rare in infants, in whom acute simple adenitis is, however, not uncommon. Tuberculous adenitis is most frequently encountered during the ages of from three to ten years and it is usually primary in nature, infection occurring by way of the tonsils and resulting from exposure to an individual with open tuberculosis or from drinking milk from a tuberculous cow. There is no doubt about milk infection being a frequent cause as the bovine type of bacillus has repeatedly been demonstrated in these glands. The child may also infect itself if it has a pulmonary tuberculosis; in such cases the adenitis is a secondary condition due to infection of the glands from sputum which is coughed up and which brings the tubercle bacilli in contact with the tonsils whence they are carried into the cervical lymph nodes.

Tuberculous cervical adenitis is usually unilateral and begins as a slowly increasing enlargement of one or more lymph-nodes which at first are painless and attract little attention. The gland gradually becomes larger and the swelling shows periods of exacerbation during which it may become sensitive. If the process continues the overlying skin eventually reddens, the gland which at first was firm and elastic in feel shows evidence of softening and breaks down. Spontaneous rupture takes place in due time with the formation of a sinus with ragged edges, infection of the skin and surrounding tissues and slow healing, leaving an unsightly scar.

A single gland may be affected but more frequently there are a number of glands involved at the same time. In the early stages the glands remain discreet and they are freely movable while the overlying skin remains loose and unattached to the glands. In the later stages, however, the glands tend to fuse, forming a hard, nodular mass to which the overlying skin becomes attached. When such a mass breaks down several points of softening can be detected as a rule and a single opening does not suffice to satisfactorily drain the mass. The course of tuberculous adenitis is chronic, usually extending over a period of months and sometimes years. Resolution may occur, but the chances are against such an outcome, unless early treatment is instituted.

The *treatment* of tuberculous adenitis is first of all the removal of diseased tonsils or teeth in order to stop any further re-infection of the glands in question. Constitutional treatment is also important, fresh air and cod liver oil being especially useful. The seashore is usually of more benefit than the mountains. In the early stages *calcarea carb.* is the most useful remedy. *Mercurius* and *hepar sulph.* are better indicated in simple acute adenitis. Some cases especially those of a very sluggish type appear to be benefited by *tuberculin* in potency.

X-ray treatment has given excellent results in many of my

cases and should be tried whenever the case is seen early. As soon as signs of break-down appear the gland should be opened, all necrotic tissue removed and the wound packed with iodoform gauze and allowed to heal by granulation. The radical removal of tuberculous glands is advocated by some surgeons but while this procedure undoubtedly gives excellent results in selected cases, nevertheless the more conservative method of waiting for signs of necrosis and then promptly draining the gland is the best routine method to adopt. The treatment of the other clinical forms of tuberculosis is discussed under their proper headings.

HEREDITARY SYPHILIS.

Syphilis in childhood is almost invariably an inherited disease, although it may be acquired during parturition from a primary lesion of the vulva or from subsequent exposure to infection. This is usually the case when the mother acquires syphilis late in pregnancy, for if the disease is acquired after the eighth month the child escapes direct placental infection. Infection of the fetus takes place through the placenta. There is probably no direct transmission of syphilis from the father to the ovum as was formerly supposed, but the mother is infected primarily whether she shows clinical evidence of syphilis or not, and the spirochetæ are first carried from the maternal blood to the placenta. As a result of the infection of the placenta its blood vessels become diseased and the spirochetæ gain entrance into the circulation of the fetus. Fetal syphilis is, therefore, primarily a general blood infection and the newborn may present no characteristic anatomical lesions. The spirochetæ can however be demonstrated in the organs in such cases.

Acquired syphilis differs from the above forms both in the manner in which the disease gains access into the system and in the presence of the primary sore, or chancre, which is never found in inherited syphilis.

The fetus has very little resistance to the invasion of the syphilitic virus for which reason the lesions eventually become very wide spread, and it does not possess the power of developing anti-bodies so that the Wassermann reaction is usually absent in young infants.

It was formerly held that mothers of syphilitic infants who did not present clinical evidence of syphilis were immune to the disease. Colles made the observation that such mothers could nurse their infected offspring without developing a primary lesion of the nipple or subsequent indications of infection. This became known as "Colles' Law." The explanation of this apparent contradiction to the ordinary phenomenon of syphilis, lies in the fact that these mothers are syphilitic as proven by the large percentage of positive Wassermann reactions obtained from such women.

Early or precocious hereditary syphilis may result in miscarriage. Children showing active signs of syphilis at birth are seldom born alive. They may appear macerated, or the body be covered with an extensive bullous eruption.

The variety of hereditary syphilis described as *syphilis hereditaria tarda* by Fournier, in which the appearance of specific lesions is supposed to be delayed until after the third year of life, is not recognized by most syphilographers, it being held that the early manifestations in these cases were overlooked. Again, symptoms occurring in later childhood may be the result of an innocent infection (*syphilis insontium*).

The *pathological lesions* of hereditary syphilis are well developed in most of the internal organs. The lungs show an increase in the interalveolar connective tissue and proliferation of the alveolar epithelium (*pneumonia alba*). The liver may be enlarged as a result of round-cell infiltration of the inter-acinous spaces and pericellular cirrhosis; there may be gummata (rare) or simple interstitial connective tissue proliferation. These changes begin in the periportal region and spread into the acini, invading them with new connective tissue

and blood-vessels. The spleen is enlarged as a result of diffuse cellular infiltration of its interstitial tissue.

In the bones, epiphysitis is a characteristic change already observed in the fetus. Other conditions will be referred to under the clinical manifestations of the disease.

Symptoms.—The classical manifestations of syphilis which develop after birth are palmo-plantar pemphigus, coryza, cutaneous syphilides, epiphysitis and cachexia. These symptoms develop in the order named, and it is important from the standpoint of diagnosis and treatment to have a proper understanding of the evolution of the disease.

Pemphigus may develop in the fetus during the sixth or seventh month. It is, therefore, usually present at birth and represents the earliest clinical manifestation of syphilis. The lesions consist of bullæ about one centimeter in diameter, situated upon the palms of the hands and soles of the feet. The epidermis is loosened from the true skin and has a bleached macerated appearance. While the bullæ are intact they contain a yellowish fluid. The bullæ soon dry up and the epidermis falls away, leaving a raw copper colored surface.

Coryza is one of the most constant manifestations of syphilis. At times it is the only symptom present the child appearing to be in good health otherwise.

The earliest manifestations are a serous nasal discharge which later becomes sero-purulent. The nose is obstructed and the infant's respirations become noisy and embarrassed. The discharge is irritating and leads to erosion of the skin about the nares and the mouth with the development of fissures in these localities.

Cutaneous syphilides appear shortly after the coryza. They consist of pink, oval macules more or less general in distribution. At the end of a few days they become copper colored and desquamate. Wherever these papules are exposed to moisture, especially about the buttocks and about the mouth their surface becomes macerated and they increase in size and be-

come converted into ulcers and fissures. In severe cases the infants are emaciated and present bullous lesions on the palms of the hands and the soles of the feet at birth. This is soon followed by the development of diffuse infiltration of the skin with a tendency to scale; pustules; ulcerating lesions of the mucous membranes. In less virulent cases there appear at the end of a few weeks macular syphilides on the lower portion of the abdomen and on the buttocks; papules and pustules may coexist. The pustules are especially common upon the face and buttocks. They have a tendency to ulcerate deeply forming dark-colored crusts. The skin appears shrivelled, poorly nourished, and presents a brownish discoloration. Associated symptoms are hoarse, plaintive cry, mucous patches in the mouth, rhagades at the angles of the mouth, anal condylomata and gastro-enteric catarrh, inducing loose, green stools. The syphilitic child soon develops malnutrition and a severe secondary anemia; the face wears a characteristic old and anxious expression. The internal organs, as mentioned above, are the seat of diffuse interstitial hyperplasia of the connective tissue, with resulting degenerative changes in the parenchyma of the liver, lungs and digestive system. Enlarged spleen in an infant under three months old is strong presumptive evidence of syphilis. The visceral lesions are responsible for the malnutrition and eventual death of the syphilitic infant, although it may die from a basilar meningitis.

The later manifestations of syphilis, occurring in cases not so malignant from the outset, are those referable to the bones, teeth, organs of special sense and nervous system.

In the osseous system epiphyseal osteochondritis and dactylitis may occur early in the disease. Osteochondritis develops at the epiphyses of the long bones and by interfering with the growth of the bone may lead to deformity. The symptoms of epiphysitis are acute and simulate arthritis. The child holds the limb as if paralyzed on account of the pain. The lower end of the humerus is most frequently involved. Dactylitis pre-

sents a characteristic fusiform swelling of the fingers also attacking the metacarpal and metatarsal bones. Ulceration often results with the destruction of the bone and integument. Osteoperiostitis of the tibia, resulting in rounding out of the tibial crest and curving of the shaft—the sabre-blade deformity—is very characteristic of hereditary syphilis. In rickets the sharp crest of the tibia remains unchanged, while deformities of the bone are most marked at its lower end. Cranial exostoses upon the frontal and parietal bones are also found in well-developed cases.

The milk teeth are delayed and decay early; the permanent teeth present pathognomonic signs first described by Jonathan Hutchinson, for which reason they are known as Hutchinson's teeth. The upper central incisors are dwarfed and present a notch upon their cutting surface, while the other teeth are poorly developed.

Two other conditions to which Hutchinson has given much prominence are interstitial keratitis and otorrhea. Chronic otorrhea or sudden deafness should always arouse a suspicion of syphilis. Interstitial keratitis is a frequent symptom of syphilis, developing at the time of puberty.

Nasal deformity is a characteristic sign of hereditary syphilis as well as radiating linear scars at the angles of the mouth. The latter results from ulcerating mucous patches, while the former is due to diffuse gummatous rhinitis, with accompanying ozena.

Gummatous infiltration of the brain and cord may lead to a variety of disturbances in the nervous system. Meningitis; epilepsy; dementia paralytica; tabes dorsalis and hydrocephalus are among the most important nervous affections that can be traced frequently to a syphilitic origin.

The *diagnosis* of syphilis is not difficult in the presence of a clear family history and clean-cut consecutive manifestations of the disease, but it may present difficulties when isolated symptoms are encountered. In the first place, a history of

miscarriages in the mother followed by the birth of a still-born infant or one that died of "inanition" in early infancy is strong presumptive evidence of syphilis. Secondly, the presence of snuffles at birth is an important symptom. This, however, must not be confused with the innocent snuffles present at birth or due to adenoids. Specific snuffles begins in the second or third week and is associated with crust formation in the nose and often saddle nose. It gets progressively worse. Other suggestive symptoms are the malnutrition; the hoarse cry; enlargement of the spleen; mild grade of hydrocephalus; buccal and anal ulceration and papulosquamous lesions in the palms of the hands and soles of the feet. A Wassermann reaction can usually be obtained in older infants but in the first weeks of life it is absent. The same can be said of Noguchi's lutein skin reaction. Many cases of congenital debility and hemorrhagic disease of the newborn are presumably syphilitic because a positive Wassermann reaction can be obtained in the mother although absent in the infant.

The later manifestations of syphilis are all characteristic, and in the presence of such symptoms as Hutchinson's teeth; radiating linear scars; flattened nose-bridge; dactylitis and interstitial keratitis there should be no doubt of the diagnosis. The Wassermann reaction is as reliable at this age as in adults.

Prognosis.—The death rate among syphilitic infants is high and the prognosis depends upon the severity of the early manifestations to a great extent. When symptoms are discovered at birth or shortly after, the infant usually succumbs because of the presence of an extensive general infection with very little natural resistance. When the early symptoms are mild and their appearance is delayed, the outlook is better. If an infant is well nourished at birth and can be nursed by its mother there is always a chance of eradicating the disease with proper treatment. A bottle-fed infant, however, has a decidedly poorer chance of recovery. The first born of a syphilitic parent usually presents the disease in a more serious form than the subsequent children, and is more likely to succumb.

Treatment.—The syphilitic infant is a menace to its surroundings, for, with the exception of its mother, it is capable of infecting anyone with the disease. The lesions in the mouth and the discharges from the nose or from ulcerating papules or pustules anywhere upon the body are the sources from which infection may take place.

Mercury is the best remedy with which to begin the treatment as it corresponds to the majority of the symptoms of secondary syphilis, the stage in which hereditary syphilis first manifests itself. The preparation from which I have obtained the best results is the *protoiodide*, in doses of two to three grains of the second decimal trituration four times daily. When the case is one of unusual severity with early appearance of active manifestations, the action of the *mercury* is more prompt and less likely to cause intestinal disturbances if given by inunction instead of by mouth. Ten grains of mercurial ointment, diluted with cold cream, should be rubbed into the abdomen, thighs or axilla daily until the symptoms have been brought under control. In the late manifestations of hereditary syphilis the *iodide of potash* must frequently be employed in material doses in conjunction with *mercury*.

Cases which do not promptly respond to mercurial treatment, or in the more severe cases presenting such conditions as destructive changes in the nasal septum, rapidly progressing inanition, involvement of the eyes or of the nervous system, recourse should be had to the more quickly acting arsenical preparations. *Neosalvarsan* is the best one of these because it is least toxic and irritating and may be given intramuscularly in oil. The best results, however, are obtained by giving *salvarsan* intravenously.

The superior longitudinal sinus has been used for the purpose of administering *salvarsan* intravenously to an infant. Helmholtz, of Chicago, in 1915 called attention to the clinical practicability of this route. Dunn and Howell, of Boston, successfully used the sinus for obtaining blood for the Wassermann reaction

and later gave *salvarsan* injections into the sinus. This procedure is, however, not without danger and intravenous or intramuscular injections are, therefore, to be recommended.

The dose of *salvarsan* is .05 grams for an infant one to three months old and 0.1 gram for three to six months. Holt allows 0.01 gm. per kilogram of body weight. The dose of *neosalvarsan* is 1½ times that of *salvarsan*. The injection may have to be repeated at the end of a week.

As soon as the urgent symptoms have been controlled mercurial treatment should be resumed. The Wassermann reaction may be employed in order to control the treatment, but it must be remembered that the reaction is often absent in syphilitic infants during the first month.

The following remedies are mentioned in the homeopathic literature on the treatment of syphilis and should be consulted for special symptoms and conditions:

Aurum.—Tertiary manifestations; exostoses on skull, tibia and bones of forearm; dactylitis with ulceration; caries of nasal bones; defective development of genital organs; infantilism; mental depression.

Kali bichromicum.—Snuffles; harsh voice and hoarse cry; deep ulcers on the edge of the tongue; ulcers on the velum palati eating through; ulceration of nasal septum (cartilaginous portion); ulcers in general, with characteristic punched-out appearance.

Kali hydroj.—Tertiary syphilis; diffuse and circumscribed gummatous infiltrations; mercurialization; interstitial keratitis; otorrhea; swelling and ulcerative destruction of uvula.

Mercurius.—The homeopathic relationship of *mercury* to certain stages of syphilis is a firmly established fact. An analysis of the cases successfully treated with *mercury* indicates that its most marked effects are the healing of ulcers and improvement in the general health, both of which belong to the truly homeopathic action of the drug (Hughes, *Pharmacodynamics*). Its "tonic" action is due to its hematic power,

while its control over diffuse inflammation and swelling of the mucous membranes, accompanied by ulceration and inflammations of serous membranes, periosteum and skin, depends upon its specific action upon these structures. This primary, specific action covers almost completely the early manifestations of hereditary syphilis, and the manifestations of mercurial poisoning cover many of the destructive manifestations of the disease. Impetigo and rupia, rapid ulceration of the mucous membranes, skin and bones etc., strongly call for *mercury*, especially in combination with *iodine*, as recommended above or in larger doses when symptoms become urgent (inunctions).

Nitric acid.—Deep, irregular ulcers on border of tongue, upon tonsils and soft palate; sticking pains in ulcers; rhagaded at angles of mouth; pustular and squamous syphilides; mercurial stomatitis and cachexia; urine strong, ammoniacal; condylomata.

Sulphur.—Syphilitic children often require an occasional dose of *sulphur*. The symptomatology of this remedy is too extensive to be considered here, its sphere of action embracing both general and special indications. *Psorinum* may likewise be called for occasionally.

Thuja.—Flat, condylomatous lesions about the anus and ulcerating papules on the scrotum.

RHEUMATISM.

While rheumatism in the adult is usually seen as an acute polyarthrititis of acute onset and limited duration, in childhood it presents the characteristics of a subacute generalized infection with less tendency to affect the joints but with an almost universal one to attack the valves of the heart. The etiological relationship between the tonsils and rheumatism is much more clearly evident in children than in adults and in many cases the onset can be traced directly to an attack of tonsillitis. Rheumatism is rarely seen in children under four years of age.

Acute arthritis, multiple in character and usually affecting

the smaller joints, namely the wrists, ankles, and elbows, is the most striking and characteristic symptom of rheumatism. Arthritis of a single joint, particularly one of the larger joints, is, however, rarely due to rheumatism; sepsis or tuberculosis should always be suspected in such cases. The fingers are sometimes involved and this is indicative of a more severe type of rheumatic infection.

Rheumatic nodules are an unusual manifestation of rheumatism and they are found in a minority of cases as small oval fibrinous nodes situated along the tendons of the fingers, at the back of the elbow, about the knee joints and in other localities. While they are characteristic of rheumatism, still they are not found frequently enough to be of much diagnostic value.

"*Growing pains*" are at times an indication of rheumatism and when a child complains of them it should be carefully examined for evidence of arthritis, muscular spasm and heart disease and the temperature should be taken regularly to determine the presence of a slight continued fever. Frequently the so-called growing pains are only muscular soreness which comes from over-exertion or fatigue or the pains have resulted from a sprain. Torticollis is the most frequent form of acute rheumatic myositis seen in childhood.

Chorea is so frequently associated with rheumatic arthritis or with rheumatic heart disease that the relationship between it and rheumatism cannot be questioned. Chorea frequently follows in the course of an arthritis and develops endocarditis as a complication. There is no doubt that in such cases the rheumatic toxin is the cause of the nervous symptoms. Chorea may develop independently of rheumatism but such cases are very likely toxic and may be more closely related to the rheumatic cases than is apparent.

Endocarditis is by far the most important and perhaps the most frequent complication of rheumatism in childhood. It is really a question whether we can call it a complication as it appears to be the primary manifestation of the infection in

many instances, ante-dating the occurrence of arthritis. It is an interesting clinical observation that cases with marked joint involvement often escape heart involvement while cases with endocarditis and pericarditis may present few if any joint symptoms. Endocarditis usually runs a subacute course and may become chronic; fever often persists for months. Even after the fever has subsided there is a strong tendency for relapses to occur. A leucocytosis is usually present while the fever persists.

A number of *skin* manifestations have been observed associated with rheumatic infection. They belong to the erythema and hemorrhagic group and present a multiplicity of clinical features.

Diagnosis.—Symptoms resembling rheumatism occurring in a child under four years of age should always be investigated carefully before a diagnosis is made. Pains about a joint or a fixed joint do not necessarily mean an arthritis. The conditions most likely to be confused with arthritis are epiphysitis and infantile scurvy.

Epiphysitis is a manifestation of congenital syphilis and other signs of syphilis are usually present. It is generally found in infants under six months old, affects by choice the epiphysis of the humerus causing a pseudo-paralysis of the arm with tenderness and slight swelling above the joint. The onset is slow and the course chronic.

Infantile scurvy occurs in the latter part of the first year, affects principally the lower extremities and careful examination reveals tenderness and swelling in the lower end of the long bones and not in joints. Swollen, bleeding gums are associated.

Arthritis in infants is usually septic or gonorrheal; in the latter instance it complicates gonorrheal ophthalmia or vulvovaginitis. Aspiration of a joint reveals the presence of pus.

Tubercular arthritis is of gradual onset, chronic in course and is usually monoarticular, involving one of the larger joints.

These are the hip, knee and shoulder, given in the order of their frequency. The cutaneous tuberculin test is usually positive and the X-ray shows evidence of bone destruction.

A primary *endocarditis* should be considered rheumatic if any other cause for the same can be excluded. As a rule the history reveals a former attack of arthritis or chorea or these conditions develop after the endocarditis.

Treatment.—The most important therapeutic measure in the treatment of rheumatism is prophylaxis. There is every reason to believe that our first duty to a child which presents rheumatic symptoms is to make a searching investigation for any evidence of a focal infection. Carious teeth and infected gums should receive immediate attention. Enlarged infected tonsils or small, buried tonsils with palpable sub-tonsillar nodes should be removed. The nose and post-nasal space should also receive attention as infected adenoid tissue or a sinus infection may be present.

General hygienic measures which aim to increase the child's resistance to infection must be enforced. Woolen underwear in the winter is usually recommended but this may be objectionable in some cases. A carefully instituted hardening process, accustoming the child to sleep in the open air; avoiding over-heated apartments; cold sponge baths; strict attention to the digestion and bowels and a rest hour during the day are the most important general measures to be observed. Rheumatic children should be strictly guarded from others with colds, sore throat and the infectious diseases. In a minor percentage of cases removal to a warm, equable climate in winter may become necessary. The diet need not be restricted in nitrogenous food to the extent which is often done; there is no relationship between meat or sugar in the diet and rheumatism as in the case of gout. The diet however should be a simple, nutritious, well-balanced one in which milk, butter, eggs, bread, cereals, green vegetables and fruit should play the most important role. During acute attacks a low diet is indicated. For

the anemia which is a result of rheumatic infection iron preparations and cod liver oil are indicated.

The *remedies* which are found useful for the general and arthritic symptoms in adults are of equal value in the child. When suffering is extreme the pain must be relieved. The snug wrapping of a joint in cotton or its complete immobilization often gives sufficient relief. *Salicylates*, however, may be necessary to allay the pain. Unfortunately they do not influence the infection or prevent recurrences and complications; they are purely palliative. The average dose for a child is a five grain powder of equal parts *sodium salicylate* and *sodium bicarbonate* given with plenty of water every three to four hours.

Every case of rheumatic infection running a temperature, whether arthritic or choreic in type, should be kept in bed for observation until the temperature has remained normal for several days. Should any evidence of an endocarditis appear, rest in bed must be enforced until we can make certain, as far as that is possible, that the heart infection has subsided. To determine this point, the presence of fever and a leucocytosis are our chief guides.

CHAPTER XVII.

ACUTE INFECTIOUS DISEASES.

EXANTHEMATA.

The exanthemata constitute a group of acute infectious fevers usually occurring epidemically and characterized by the eruption of an exanthem upon the surface of the body. The group is composed of measles, rubella and scarlet fever. They are all contagious, but scarlet fever is less so than the others, perhaps because individual susceptibility to it is not as universal as with measles. Whether the exanthemata are due to bacteria or to a protozoon has not yet been determined.

Space forbids reviewing the experimental work which has been done in order to determine the etiological factors in measles and scarlet fever. Suffice it to say that up to the present time the causative agent of neither of these contagious diseases has been isolated. It has been possible to transmit measles to monkeys by inoculations with the blood and nasal secretion of patients suffering from the disease. The contagium is a *filterable virus* which resists drying but is destroyed by a temperature of 55° C. In scarlet fever streptococci are persistently found in the throat and in the blood in the majority of severe and fatal cases; however the role of the streptococcus is more likely that of a secondary invader than an etiological factor.

MEASLES, RUBEOLA.

Measles is one of the commonest of the acute infectious diseases of childhood and there appears to be a universal susceptibility to it. A child that has been exposed to measles rarely escapes contracting the same. It occurs most commonly in epidemics during the months favoring catarrhal affections; spring epidemics are usually the severest. One attack affords immunity against another. The period of incubation is from

ten days to two weeks in the average case. Contagiousness is present from the time of invasion, being most pronounced at the height of the catarrhal manifestations and fever. It rapidly vanishes with the disappearance of the eruption, and at the end of the third week there remains little or no danger of contagion. The contagion is usually spread by close contact, and is seldom conveyed by means of intermediate objects or a third person, it also being readily destroyed by thorough airing and fumigation. Measles, however, is more readily disseminated than scarlet fever or diphtheria, and an epidemic is more likely to attain wide-spread proportions than in those diseases.

Symptoms.—The course of a typical case of measles is in three stages. These are characteristic of the exanthemata in general, but most clearly defined in measles. They are: the prodromal stage, the stage of eruption, and the stage of desquamation.

The first stage is characterized by fever and catarrhal symptoms of gradual onset. There are bloodshot eyes and lachrymation, accompanied by chilliness and headache. The catarrhal process extends to the larynx and trachea, producing the characteristic hoarse cough. On the third day single, lentil-sized red spots are seen upon the roof of the mouth and soft palate, frequently being observed twenty-four hours before the eruption upon the skin makes its appearance. Koplik's sign appears even earlier and is more truly pathognomonic of measles in the period of invasion. Koplik describes this buccal enanthem as follows: "If we look into the mouth at this period we see in a strong light the usual redness of the fauces, perhaps not in all cases a few red spots on the soft palate. On the mucous membrane lining the cheeks and lips (buccal mucous membrane) we see a distinct and pathognomonic eruption. This consists of small irregular spots of bright-red color; in the centre of each spot is the interesting sign to which I wish to call attention. In strong daylight we see a most minute bluish-

white speck. These minute bluish-white specks in the centre of a reddish spot are absolutely pathognomonic of beginning measles. This sign is present in all cases twenty-four hours before the skin eruption, and often three days preceding it."

The second stage begins on the fourth or fifth day. The eruption makes its appearance first on the face, the earliest spots occurring at the border of the scalp and behind the ears. Thence it spreads over the entire body surface, the eruption being completed in two to three days. Its spread, however, may be irregular and interrupted and desquamation may occur on one portion of the body while the eruption is appearing on another. The exanthem is the product of a superficial dermatitis, with papule formation, resulting from round-cell infiltration about the papillæ, the cutaneous glands and small blood vessels. There may also be edema of the skin accompanying the inflammatory process; this is most prominently seen upon the face. The eruption proper consists of numerous roundish, lentil-sized red spots, slightly raised above the level of the surrounding skin, or containing in their centre a little papule. Where they are very numerous they coalesce, forming crescentic plaques, or they may fuse into large, spotted areas. Cases in which the hyperemia is so great as to cause cutaneous hemorrhages are described as petechial, or black measles; in these cases the eruption assumes a dark color from petechial hemorrhages. Petechial measles is by no means always a more serious condition than the ordinary form; in fact, I have seen a number of cases running a rather mild course, in which the eruption assumed this hemorrhagic type.

In young children convulsions sometimes occur at the time the eruption makes its appearance. The catarrhal symptoms reach their acme, and bronchopneumonia and troublesome diarrhea are to be feared during this period. Catarrhal inflammation of the conjunctiva, nose, pharynx, trachea and bronchi are so closely associated with the course of an attack of measles that they are really to be looked upon as characteristic lesions

of the disease. The strong tendency for the process to extend from the bronchi into the bronchioles and air vesicles is one of the most dangerous features of measles, and almost every fatal case is directly due to bronchopneumonia.

The inflammation of the pharynx and larynx may become croupous, and suppurative otitis media may appear as a complication at this stage, although neither of these conditions are as common in measles as in scarlet fever.

In the alimentary tract a similar catarrhal condition may become established, showing itself as anorexia, vomiting, heavily coated tongue with enlarged marginal papillæ, and diarrhea. The latter, when once established, is liable to continue throughout convalescence.

At the end of about four days the eruption begins to fade, disappearing first on those localities where it was primarily seen. In mild cases it has already become much paler at the end of twenty-four hours, and it may disappear entirely from one part while another part is being invaded. With the fading of the rash desquamation takes place in the nature of fine, branny scales, first noticed upon the face and neck. It is completed in a week in the average case, seldom continuing beyond this time.

The eruptive period is prolonged in those cases in which it becomes hemorrhagic. Here it assumes a deep-red color, gradually becoming darker (ecchymotic) and slowly fading out as the blood-pigment is absorbed. Again, the eruption may suddenly disappear, indicating circulatory failure.

The temperature is highest during the eruptive period, when it may reach 104° F. for two or three days. The average duration of fever is about a week. In a typical case there is an abrupt rise at the time of invasion—about 102.5° F. (initial fever). It soon falls to a lower point, gradually rising again until the fourth day, when the eruption makes its appearance. At this stage it may reach 104° F. and higher. It may drop by crisis on the fifth day or there may be a gradual decline so that normal is not reached until the seventh day. A longer

febrile period or an accession of fever during the period when it should normally decline, indicates a complication.

Among the many complications liable to arise during the course of measles or appear as sequelæ, the following are the most important and most frequent; bronchopneumonia (children under three years); pleuropneumonia and empyema (three years and over); membranous croup; putrid sore throat; noma; entero-colitis; conjunctivitis; keratitis; otitis media.

The frequency with which tuberculosis develops after measles is noteworthy. In some instances latent scrofulous lesions are fanned into activity, while in others a primary pulmonary infection apparently occurs. The congestion of the bronchial glands which accompanies measles, renders them more liable to infection with the tubercle bacillus. According to Osler, tuberculosis is the most important sequela—either an involvement of the bronchial glands, a miliary tuberculosis, or a tuberculous bronchopneumonia. The observation that the von Pirquet cutaneous tuberculin reaction becomes negative during an attack of measles in children who have previously shown a positive reaction, would indicate that the defensive mechanism against tuberculosis is temporarily abolished by the infection.

The blood in measles shows a mild degree of anemia and instead of a leucocytosis there is a lymphocytic leucopenia in the early stages. This has also been observed during the incubation period. The urine may give the diazo-reaction, but albuminuria is rare.

Treatment.—The child should be put to bed in a well-ventilated room as soon as the disease is suspected, and a temperature of 65° F. maintained if possible. It is unnecessary to render the room dark and cheerless, but the eyes should be protected from direct bright light. The child should be kept in bed until every trace of the rash has disappeared, which usually takes a week. He should not be permitted to go out of doors until he has been up for a week, but during this time

he may be given a daily indoor airing, the windows being widely opened and the child clad as for outdoors. After sixteen days have elapsed from the time of onset of the disease, the quarantine may be lifted.

The removal of the branny scales of epidermis is greatly facilitated by rubbing the child with olive oil, followed by sponging with tepid water and Castile soap. This measure should be employed for several evenings in succession after the febrile symptoms have abated. During the febrile period there is no objection to the cleansing sponge-bath of tepid water. If conjunctivitis be present the eyes should be flushed several times daily with a 2 per cent boric acid solution.

In cases in which the rash is tardy in coming out, or in which there is a recession of the same, a warm bath or pack is of great service. With recession of the rash the condition often becomes grave. When due to cardiac failure, stimulation is indicated, and a warm bath is a valuable adjuvant when serious congestion of internal organs (bronchopneumonia, meningitis, etc.) exists as a complication.

In feeding cases of measles we must bear in mind the tendency to diarrhea, just as in scarlet fever we must anticipate nephritis.

During convalescence the diet should be highly nutritious, consisting largely of milk, eggs, fresh vegetables, lamb chops, etc. If a tendency to tuberculosis exists, cod liver oil may be added with advantage.

Aconite corresponds to all of the early symptoms of a typical case of measles and it may be looked upon as a specific. As a rule, it is the only remedy required unless complications occur.

Arsenicum is indicated in adynamic cases in which there is pronounced prostration; scanty rash from circulatory failure or hemorrhagic form of eruption; anxiety and restlessness; albuminuria.

Bryonia.—*Bryonia* may be required for the associated

bronchitis with hard, dry, painful cough. The rash is slow in appearing, but, when once established, it is usually abundant and characteristic. The accompanying symptoms are the cough; great lassitude and irritability; anorexia, with thirst for large quantities of water; constipation. Both *bryonia* and *gelsemium* have been credited with the power of hastening the appearance of a tardy rash, but there is no clinical proof that they can produce such a result.

Coffea is recommended for the short, dry, teasing cough of measles, frequently becoming a most distressing complaint in nervous, delicate children.

Euphrasia.—This remedy is useful for the eye complications when there is a profuse corroding discharge from the eyes, with profuse, bland nasal discharge (*allium cepa* has the opposite condition).

Gelsemium.—*Gelsemium* may be indicated in place of *aconite* in cases of mild gradual onset where the fever is slight and there is slight chilliness with marked catarrhal symptoms, headache, drowsiness, photophobia and general aching as in mild influenzal attacks.

Kali bichromicum is of value for a deep, loud cough, with expectoration of stringy yellowish mucus; intense conjunctivitis, sometimes going on to keratitis and ulceration; stitches in the ears, extending into the head and neck; watery diarrhea, with tenesmus; ulcerated sore throat. Even when the symptoms are not so severe or characteristic as above stated, this remedy is frequently of great value, especially when *bryonia* does not control the bronchitis. It is followed by *pulsatilla*.

Lachesis.—Severe hemorrhagic type of the disease.

Pulsatilla may be indicated early, although its sphere of usefulness lies mostly in the clearing up of the cough and catarrhal symptoms lingering after measles. It is followed well by *hepar sulph*.

Other remedies which may be called for upon special indications are:

Belladonna in alternation with the *red iodide of mercury* may be needed when a severe angina complicates the measles. *Belladonna* is also useful for the nervous symptoms which may occur during the disease.

Hepar and *spongia* may be required when the cough becomes croupy.

Phosphorus and *antimon tart.* in those cases in which bronchopneumonia develops.

SCARLET FEVER.

(SCARLATINA; French, LA SCARLATINA; German, SCHARLACH.)

Scarlet fever is an acute eruptive fever which is highly contagious and is characterized by a sudden onset with high fever, sore throat and a scarlet rash, whence it derives its name.

Scarlet fever was known to the ancients; however to Sydenham belongs the credit of first differentiating it from measles and of writing a clear clinical description of the disease. Since the eighteenth century scarlet fever has been well-known in Europe and the first epidemic recognized in the United States occurred in Massachusetts in 1795.

Etiology.—Scarlet fever occurs in epidemics in all large communities usually in the fall and winter months. Sporadic cases may occur at any time and the disease frequently develops in conjunction with a severe burn when it develops outside of an epidemic. Scarlet fever is distinctly a disease of school age and is more prevalent among children at the time of year when they attend school than when they are living mainly out of doors and in less intimate contact. It is more prevalent in densely populated cities than in rural districts and for this reason more common in our latitude than in warmer countries. The greatest degree of susceptibility exists between the ages of two and six years; infants usually escape, especially those who are nursing at the mother's breast. If the mother has not had scarlet fever the infant is perhaps as susceptible as the older

child. After puberty susceptibility gradually decreases; many adults, however, contract scarlet fever, both in mild and severe forms. The immunity obtained from a previous attack is practically absolute.

While scarlet fever is not as contagious as measles, its spread being slower and less extensive than the latter in communities or non-isolated quarters harboring cases, still its causative agent possesses much greater tenacity to life, and is much more readily carried from one locality to another by means of a third person or by contaminated objects than measles. It retains its vitality for months, and requires active germicidal measures for the successful disinfection of infected places and articles of dress, bedding, etc.

The period of contagiousness lasts about five weeks, beginning with the invasion of the disease, reaching its height during the febrile period and persisting until desquamation is complete. The source of infection lies in the catarrhal discharges from the nose and throat and in the pus from a suppurating ear or gland. It is generally held that the scales of desquamated epidermis cast off during convalescence from the disease harbor the infective agent, but this has not been proven and is now looked upon as an improbable cause for contagion.

The exact nature of the causative agent of scarlet fever still remains obscure. Streptococci are found in the blood in a certain percentage of cases, but they are rather to be looked upon in the light of a secondary infection than as the primary cause of the disease. They occur with relatively greater frequency in the more severe and protracted cases, but they may be absent in some of the fatal cases. Mallory has demonstrated certain bodies in the skin of four cases of scarlet fever, which he considers one of the stages in the development of a protozoon. Inclusion bodies have been found in the polynuclear leucocytes by Dohle and others but they have also been found in other conditions where there was a streptococcus infection and cannot, therefore, be considered specific. The period of incubation is

short, usually less than a week, and in many cases only two to three days.

Symptoms.—The course of a typical case of scarlet fever may be divided into the stage of invasion, stage of eruption and stage of desquamation. Prodromata are rare, the invasion being abrupt, with fever, headache, vomiting and sore throat. Such a combination of symptoms occurring in a child should always lead one to suspect scarlet fever. The temperature may rise very rapidly to a high point, reaching 104° F. and over; in mild cases, however, it may rise but inconsiderably. The pulse likewise is affected in a characteristic manner, attaining a rapidity of one hundred and twenty to one hundred and thirty beats per minute quite early in the attack. The throat is highly inflamed, a diffuse erythematous blush covering the tonsils, pharynx and soft palate. Later in the disease diphtheritic patches may appear (pseudo-diphtheria).

The rash.—Within from twelve to thirty-six hours from the beginning of the fever the eruption makes its appearance, first showing itself about the neck and chest, whence it rapidly spreads over the entire body, this being accomplished within twenty-four to thirty-six hours, or in even a shorter period of time. It develops most intensely on the neck, over the extensor surface of the extremities, about the joints, and on the dorsum of the hands and feet. A peculiar pallor about the mouth is frequently seen, producing a striking contrast with the flushed cheeks, and giving rise to the characteristic “white line” of the disease. The eruption is due to intense hyperemia of the skin, accompanied by exudation of serum and round cells into the rete Malpighii, the process ending in death of the epidermis, with desquamation of variously-sized scales and flakes. The predominating feature in the pathology of the cutaneous manifestations is vascular paralysis. When typical, the rash consists of numerous, closely-aggregated red points, the size of a pin-head, evenly distributed over the entire body, giving it a bright, scarlet color. The eruptive points may be

but slightly red in the beginning, later assuming the bright, scarlet hue. The rash is more frequently a dull red than scarlet, and the general effect is produced by the erythema associated with puncta, fine vesicles and more or less goose flesh. The punctate spots are the result of inflammation around the hair follicles, and they may become large enough to impart to the skin a distinctly rough feel. The points may be flat or elevated, round or lentil-shaped, and with increasing hyperemia they become confluent, the skin becoming turgescient and tense. The swelling is most marked about the face and eyes. Another deviation from the usual eruption is the appearance of roseola-spots of various sizes and shapes, separated by pale areas of skin (*scarlatina variegata*). In some cases the rash does not become general, often being absent from the face. It may be extremely faint in color, or assume a deep purplish hue, or become hemorrhagic.

At the height of the eruption the skin is burning hot to the touch, and the patient complains of burning, stinging and itching; at this time also, all other symptoms are most intense.

Pressure with the finger causes momentary disappearance of the rash, which re-appears from the periphery toward the center, differing in this respect from the rash of measles. If the finger be drawn across the skin a pale line is temporarily caused by the pressure of the finger which soon disappears but after a few seconds it re-appears and may persist for several minutes.

Fever.—The temperature curve of scarlet fever is one of abrupt onset, the fever running high with slight remissions during the first three or four days and then gradually subsiding by lysis. The average duration is one week.

The *tongue* is heavily coated; the edges, however, remaining red. In the course of a few days the coating is shed, leaving the red and swollen papillæ exposed, with the resulting characteristic appearance described as "strawberry tongue." Enlargement of the papillæ of the tongue is such a constant symptom

of scarlet fever that it becomes a most valuable diagnostic sign. Indeed, McCollom, of Boston, looks upon this symptom, when occurring in association with fever and sore throat, as pathognomonic of scarlet fever, irrespective of the presence of a rash. In mild cases, however, the enlargement of the papillæ may fail to develop.

The *throat* presents a characteristic intense general redness with fine red points on the hard palate. In some cases the throat manifestations are intensified and patches of membrane will be seen upon the tonsils which may spread to the soft palate and adjacent parts. This complication is usually due to streptococci and clinically is a pseudo-diphtheria. True diphtheria is rare during the course of scarlet fever, and, when associated with the same, it occurs as a sequel rather than as a complication.

Otitis is a frequent complication occurring at the height of the disease, the result of an extension of infection from the post-nasal space. It usually terminates in suppuration, and is a prolific cause of the serious types of deafness occurring in childhood. When occurring during convalescence its advent is more readily anticipated, as there is recurrence of fever, with distinct earache and impairment of hearing.

Parotitis and *cellulitis* of the neck sometimes accompany the septic process in the throat. The termination of such a process is usually in suppuration. Likewise the tonsils and lymphatic glands of the neck may be involved in suppurative inflammation rendering the prognosis most unfavorable.

Synovitis of the larger joints is prevalent during some epidemics. It develops between the first and second weeks. The duration is short, never ending in suppuration. *Endocarditis* may complicate such an arthritis.

The *blood* shows a well-marked leucocytosis, the polynuclears predominating. During the second week a marked eosinophilia may develop. The more intense the infection the higher the leucocytosis. In asthenic cases, however, there may be a

failure on the part of the organism to react and in such cases a low leucocyte count offers a grave prognosis.

The *lymphatic glands*, both the subcutaneous as well as the lymphatic structures of the viscera, are involved. There is more or less general adenopathy, the cervical inguinal and axillary glands being especially affected. A *post-scarlatinal adenitis* may develop during convalescence, involving the cervical glands. There is usually a return of fever with this complication.

Post-scarlatinal nephritis is one of the most constant and most important complications of scarlet fever, occurring typically during the third week. Pathologically, it is an acute, diffuse, productive nephritis. It is a more serious condition than the simple acute degeneration or acute exudative nephritis which may occur early in the course of the fever, just as in any other acute infectious disease. There is scanty urine and general dropsy, and suppression of urine and uremia may supervene. Although the kidney is much damaged at the time, still many cases clear up completely and show no evidence of a former nephritis in later life.

Desquamation begins shortly after the rash has faded—about the end of the first week. It begins in the localities in which the rash first appeared, as scales of varying size about the neck and chest. Gradually the entire trunk is involved in the process, desquamation being completed here long before the fingers and toes have shed their epidermis. On the latter, especially where the skin is thick, the peeling process is slow, and large pieces of skin, sometimes complete casts of the fingers, are detached in the “moulting” process. In cases where desquamation is slight, it may be found characteristically by about the tenth day at the tips of the fingers. A separation of the epidermis at the edge of the nail-bed, producing the line of “subungual cleavage,” is a characteristic phenomenon.

Prognosis.—The prognosis depends to a great extent upon the character of the epidemic; the general health of the child

before the attack; the height of the fever, and the severity of the attending complications. As a rule, the disease is more likely to prove fatal if the child is very young, especially when serious throat involvement, adenitis or otitis are associated. The degree of toxemia and the state of the peripheral circulation are important prognostic indications. A livid rash or recession of the rash, indicating failing circulation, are unfavorable signs. Cases marked by sudden onset with high fever and delirium offer a grave prognosis on account of the high degree of toxemia they present. Some cases prove fatal within the first twenty-four hours before the rash appears—"malignant scarlet fever."

Nephritis is the chief danger to be feared after convalescence has been established. The scarlatinal toxin possesses a specific affinity for the parenchyma of the kidney, and no matter how mild the attack may have been, one can never feel certain that nephritis will not develop.

Convalescence is usually protracted owing to an anemia, otorrhea and nasal catarrh, adenitis, post-scarlatinal nephritis.

Diagnosis.—Scarlet fever differs from *measles* in the abruptness of its onset, the presence of sore throat and the absence of Koplik's spots and catarrhal symptoms. The desquamation in scarlet fever is also different from that observed in measles. From *rubella* it is distinguished by the sudden onset and high fever with pronounced sore throat, by the characteristic appearance of the tongue, and by the occurrence of desquamation. *Symptomatic rashes* can usually be traced to the partaking of certain articles of food or the administration of certain medicines, or to the sepsis or auto-intoxication. The rash is of short duration, sore throat is absent, and in the absence of gastric derangement the temperature is normal. Many of the infectious fevers are at times accompanied by an erythematous rash, causing considerable confusion as to the true nature of the case. All doubtful cases, however, followed by the typical desquamation and associated with albuminuria, are to be looked

upon as scarlatina. The *serum-rash* sometimes observed after the administration of antitoxin is urticarial in character, lacks the puncta and diffuse distribution of scarlet fever and the blood shows a leucopenia.

The history of the exposure to infection is an important point in atypical and incomplete cases, as is also the appearance of the tongue and the presence of general adenopathy. The presence alone of scaling is not a proof that the case is one of scarlet fever, and scaling may be more pronounced in certain cases of *desquamative scarlatiniform erythema* than in ordinary scarlet fever. The time of onset, mode of progress and its persistence are of more importance than the mere presence of scaling (Schamberg). On the other hand, in a case of scarlet fever with well-developed rash and subsequent marked desquamation, the associated conditions, namely, fever, prostration, sore throat and adenopathy, are more pronounced than in the scarlatiniform erythemata.

Treatment.—With the occurrence of suspicious symptoms the patient should be immediately isolated. From this time on until desquamation is completed, and, if practicable, until all discharges have been controlled, the child should be kept away from others to whom or through whom it may convey the contagion. Five weeks from the beginning of the attack is usually a sufficient period of quarantine, excepting in cases with an otorrhea or any other complication in which there is a purulent discharge.

The bedroom should be freely ventilated, and all unnecessary articles of furniture and hangings should be removed, but not after they have been exposed to the contagion, unless they can be immediately disinfected. A sheet wrung out of a 2 per cent solution of carbolic acid and hung in front of the door adds to the completeness of the isolation. All kitchen utensils, etc., used by the patient should be immersed in a 4 per cent solution of carbolic acid or formaldehyde for an hour before being removed from the room. They should then

be scalded, or, better still, boiled for a quarter of an hour. The nurse and the attending physician should protect their outer clothing by wearing a long, linen coat on entering the sick room, and disinfect their hands before leaving the room. All sheets, rags, articles of clothing and furniture that can be dispensed with are best burned. The room must be thoroughly fumigated after the patient has recovered, and the bed clothes and mattress sterilized.

The itching of the skin may be relieved by rubbing the body daily with cocoa butter or cocoanut oil. The inunction of fats not only relieves the itching and burning of the skin, but it also acts as a sedative and at times reduces the fever.

In case of high fever a sponge-bath of tepid water and alcohol (one part of alcohol to three of water) is of great service. In the advent of anasarca or suppression of the urine a warm pack should be used. For the angina, a spray of alcohol one part, glycerin one part and water three parts, may be used several times daily. Likewise, the nose should be kept scrupulously clean by means of douches of a normal saline solution or Dobell's solution.

The diet should be restricted to non-nitrogenous foods. Solid food, especially meat, should be prohibited until after the third week, and in case of nephritis, a milk diet must be adhered to for a still longer period.

The *remedies* of first importance in scarlet fever are the following:

Belladonna is as nearly specific to scarlet fever in its symptomatology as any remedy can possibly be. The fever, headache, sore throat and rash are all covered homeopathically by *belladonna*. It is to be questioned, however, whether *belladonna* can prevent the disease as some have claimed, but given early it can certainly modify the severity of the symptoms. When the throat symptoms become unusually severe, developing into an actual pseudo-diphtheria, *merc. iod. rubr.* should be alternated with *belladonna*.

Rhus tox is indicated in the more toxic types of scarlet fever when there is great prostration and restlessness. Also in the advent of albuminuria early and when rheumatic symptoms develop.

Arsenicum is useful when there is profound prostration, nephritis and anasarca. Petechial eruption.

Cuprum.—Sudden recession of the eruption, with occurrence of cerebral symptoms. The *acetate of copper* is generally preferred. The *arsenate of copper* should be thought of when the condition is one of uremia.

Gelsemium.—In the early stages, when there is the characteristic dulness and drowsiness; aching and prostration; soft, compressible pulse; aching in the eyes and back of head. The throat is red and feels swollen; the eyes are suffused, and the patient feels chilly, especially along the spine.

Lachesis.—Scarlatina miliaris. Eruption becoming purple and livid; desquamation delayed; hematuria (*terebinthina*); oppression when lying down; diphtheric complication; diarrhea with foul smelling stools.

Ailanthus.—Miliary rash; small, rapid pulse; the eruption becomes dark and livid; intense angina, with acrid discharge; muttering delirium followed by stupor.

Complications and Sequelæ.—Throat complications call for *phytolacca*, the various salts of *mercury*, *kali bichrom.*, *permanganate of potash*. (See diphtheria.)

Cellulitis and Parotitis.—The most important remedy for this complication is *rhus tox*. Suppuration calls for *hepar, mercurius, silica*.

Otitis.—*Bell.*, *puls.*, *rhus tox.*, *capsicum* (mastoid involvement).

Cerebral complications.—*Apis, bell., helleb., hyos., stram., sulph.*, and *zinc*.

Nephritis.—*Cantharis* is a most valuable remedy in post-scarlatinal nephritis when there is not much blood in the urine and only moderate dropsy. When the latter is pronounced

apis and *arsenicum* are of greater service. The characteristic "smoky" appearance of the urine frequently seen after scarlet fever, from the free admixture of the blood, is a strong indication for *terebinthina*. Persistent albuminuria after scarlet fever calls for *mercurius corr.*

RUBELLA.

Rubella, Röthelm, or German Measles, is a mild exanthematous disease characterized by slight fever and a macular rash, usually occurring first on the face and involving the entire body. It presents a superficial resemblance to measles in most instances. Sometimes it is confused with the milder types of scarlet fever. Complications or sequelæ are absent. It usually occurs epidemically, and one attack confers immunity but in no wise protects against measles or scarlet fever.

Nothing definite is known of its etiology. It is contagious, but less so than measles or scarlet fever; nevertheless, it may be spread by articles of clothing, etc. Infants under six months are immune. The incubation period is from two to three weeks, but it may show considerable variation in this respect.

Symptoms.—The period of invasion is short, prodromata usually being absent. Drowsiness and fever may precede the eruption for a day or more in some cases; as a rule, the rash is the first evidence of the disease. The characteristic enlargement of the posterior cervical glands which is present from the beginning is of the greatest help in the early diagnosis of rubella. The rash is first seen upon the face, from which it spreads over the entire body in the course of twenty-four hours. Although the face is the most constant site of the eruption, even when the rash is developed but partially, still the chest and back may show the first signs of eruption in exceptional cases. The duration is about three days. Often it has completely faded from the face by the time the lower extremities are involved. There is no characteristic desquamation.

In *rubella morbilliforme* there is seen a discrete, maculo-

papular rash of pale red color, the eruptive points being slightly elevated and about the size of a pin's head or larger. These lesions have a tendency to become confluent upon the face, particularly so when they are numerous.

In *rubella scarlatiniforme* the rash is of a diffuse, uniform, scarlet color, never as intense, however, as in scarlet fever, and with unmistakable evidence of the maculopapular eruptive points in various localities (on the forehead, fingers and toes, and about the wrists).

In some instances the *diagnosis* can only be made after the mild course of the disease has been noted, in conjunction with the absence of complications and sequelæ. When, however, there is an epidemic, and especially if the child has previously had one of the other exanthemata, the diagnosis presents little difficulty. The enlargement of the posterior cervical glands is the most characteristic symptom of rubella. From measles it is differentiated chiefly by the absence of catarrhal symptoms, absence of Koplik's spots and the slight fever. From scarlatina the absence of sore throat and strawberry-tongue, the rash first appearing upon the exposed portions of the body, the low temperature and absence of desquamation and nephritis readily differentiate it.

The *treatment* is symptomatic. (See treatment of Measles.)

DIPHTHERIA.

Diphtheria is a highly contagious disease characterized by the presence of an infection of the throat which is accompanied by the formation of a false membrane upon the tonsils. The membrane may spread to the adjacent structures and invade the nose, larynx and bronchi. Death may result from suffocation through the spread of the membrane into the larynx. In the majority of cases, however, constitutional symptoms, resulting from the absorption of the toxin generated by the germ which causes the disease, are of greater importance than

the mechanical effects of the membrane and most fatalities are due to toxemia.

The disease was apparently known to the ancients. In the American colonies it was noted in Boston in 1735 and in New York it was described by Dr. Samuel Bard in 1771 as "suffocative angina." The first scientific article upon diphtheria was written by Bretonneau in 1812 and the term diphtheria was coined by this author.

Membranous croup was recognized by the older clinicians and was correctly differentiated from spasmodic croup and properly treated by them with tracheotomy. This operation was first conceived by Bretonneau but its technique was finally perfected by Trousseau. In the days of Trousseau, however, the identity of diphtheria and membranous croup was not recognized and even in recent years the diphtheritic nature of membranous laryngitis was questioned by some clinicians owing to the absence of membrane in the throat and the comparatively slight constitutional symptoms which accompany it.

Etiology.—Diphtheria is distinctly a disease of childhood. Thousands of cases occur annually among children in all large cities. In order to contract the disease an individual must show susceptibility as well as come in contact with a case of diphtheria. An apparently healthy person with a natural resistance to diphtheria may, however, harbor the germs of diphtheria in his throat and so become a "carrier" and infect others who do not possess sufficient natural resistance. Individual resistance or susceptibility can be accurately demonstrated by means of the *Schick test* and from the results of the examination of large numbers of persons it has been learned that in childhood susceptibility to diphtheria is very much higher than in adults. Schick and Park have found that about 80 per cent of the newborn are immune; Park believes that this immunity is due to the infant receiving a certain amount of antitoxin in the colostrum. From the end of the first year to the end of the fifth year the percentage of positive Schick

reactions is from 50 to 65 per cent, the highest figures occurring from two to four years. In adults there is a susceptibility of only about 25 per cent. Age and exposure, therefore, are the chief etiological factors in diphtheria. A child in the early stage of diphtheria may spread the disease to others by droplet infection during coughing and sneezing or he may transmit it indirectly by means of toys, pencils, eating utensils, handkerchiefs, etc. This mode of infection is more likely to take place during convalescence especially from mild cases which are not recognized as diphtheria and properly isolated. Like many other infectious diseases it may also be spread by means of a contaminated milk supply. Cats have been blamed as carriers but this has not been proven.

Diphtheria is endemic in all large cities but there are always periods of outbreaks affecting many children at a time. While in the rural districts and in communities without strict health supervision epidemics still attain considerable magnitude, this seldom occurs in large cities. Schools are closed temporarily if necessary and immunizing doses of antitoxin are administered gratis by Boards of Health when the expense for such a procedure cannot be borne by the parents and in this manner outbreaks of the disease are soon controlled. The medical inspection of school children has been a strong factor in the control of diphtheria. Unfortunately, however, many mild cases still go unrecognized and untreated and make the complete control of the disease a difficult problem.

The *Klebs-Loeffler* bacillus, is the etiological factor in diphtheria. This is a bacillus varying in size, being broad, straight or slightly curved and presenting a club-like extremity. It contains highly refractile, oval bodies which take the stain more deeply than the bacillus itself. The best stain for bringing out these bodies is an acidulated solution of methylene blue; a counter-stain of aqueous Bismark brown is used to stain the body of the bacillus. This is known as Neisser's stain and the most characteristic results are obtained in young cultures grown

from six to twelve hours. The bacillus grows readily upon Loeffler's blood-serum at a temperature of from 80° to 100° F., and it is therefore a simple matter to make a culture from a suspected case of diphtheria. The examination of smears from the throat is not accurate; Vincent's angina, however, may be differentiated from diphtheria by this procedure.

Pathology.—Diphtheritic membrane is most frequently found on the tonsils, soft palate, pharynx and larynx in fatal cases. It may extend into the nose or down into the trachea. The membrane which forms on mucous membrane covered with squamous epithelium such as the nose and throat is more firmly attached than that formed in the trachea which is lined with columnar epithelium. The color and consistency depend upon the amount of fibrin present, the admixture of blood and the presence of a mixed infection.

The membrane consists of a dense network of fibrin in the meshes of which pus cells, epithelial cells and bacteria are seen. The *Klebs-Loeffler* bacillus can be demonstrated on the surface and at the periphery of the membrane. The mucous membrane underlying and adjacent to the pseudo-membrane is found in inflammatory reaction with a distinct zone of hyperemia at the periphery of the false membrane. When the latter is roughly detached a bleeding surface will be left.

The lymphatic glands of the neck are markedly swollen, but do not break down. The surrounding structure may present a puffy appearance. The glandular enlargement is most marked in cases complicated by an invasion of the posterior nares. When the process is confined to the larynx there is but slight glandular involvement.

The toxemia which is associated with diphtheria produces pathological changes widely distributed throughout the body. Parenchymatous degeneration of the heart, kidneys and liver are the changes observed in the internal organs. A secondary bronchopneumonia is rarely absent in severe and fatal cases.

In such cases the Klebs-Loeffler bacillus is usually found in the lungs. In sixty-two cases of bronchopneumonia, associated with diphtheria, reported by Pearce, (*Jour. Bost. Soc. Med. Sciences*, June, 1897) the bacillus was present in fifty-two instances, being the only organism present in seventeen cases. The changes occurring in the nervous system are parenchymatous degeneration of the myelin sheath of the nerves (multiple neuritis) and at times degenerative changes in the ganglion cells of the cord.

Symptoms.—The onset is not characteristic as in most other acute infectious diseases. The chief complaint is sore throat and malaise and although the child is feverish it may not feel sick enough to go to bed. If the throat is examined at this time it will be noted that the color is of a deep red and a small grayish patch will be found on one of the tonsils. The breath is offensive and the sub-tonsillar lymph node is enlarged. If prompt treatment is instituted at this stage of the disease the temperature soon falls to normal and the spread of the membrane is arrested. It is loosened from its attachment to the mucous membrane and promptly disintegrates. If, however, the disease is not recognized and treatment is delayed there is a rapid spread of the membrane to the pillars of the pharynx and soft palate and the opposite side becomes infected. The adenopathy increases and fever and toxemia become more pronounced.

The membrane is of a grayish or yellowish color, and is firmly adherent to the subjacent mucous membrane. It has a sharply defined border and is surrounded by a red zone of hyperemia. The membrane cannot be detached without injury to the underlying structure. When antitoxin is promptly administered it frequently curls up at the edge and spontaneously separates from the mucosa. More or less edema of the soft palate is usually present. Instead of beginning as a single patch, there may be seen isolated dead-white spots of varying size upon one of the tonsils. Usually they unite into one large,

irregular patch, and the opposite tonsil soon develops a similar membrane.

In a steadily progressive (untreated) case the above distribution of the membrane will have been completed in about three to four days from the time of onset. At this time the membrane can be studied in various stages of development. At the site of origin it will be found to have attained considerable thickness, being of a brownish or dirty grayish color, with a well-defined outline and areola, and a thick, partly detached border, while in another direction it fades out into a thin, grayish film, which is invading new territory. This film likewise thickens and assumes the same color as the other portion of the membrane.

By the fifth or sixth day, in such cases, the process has reached its acme, and in a few days the membrane separates spontaneously. A red areola of reactionary inflammation is seen about its border, and it gradually loosens and comes away in pieces, leaving behind a reddened, slightly swollen and readily bleeding mucous membrane. The patient has recovered from his throat infection but there has been so great an absorption of toxin that diphtheritic paralysis with probably heart failure or respiratory paralysis are to be anticipated.

The general symptoms which are observed in conjunction with the severe throat manifestations just described are gradually increasing prostration; a rising temperature and pulse rate; offensive breath; difficulty in swallowing; nasal voice and pronounced cervical adenopathy.

The *heart* is affected by the toxin of diphtheria from the inception of the disease. The pulse is rapid from the time of onset and its rate is disproportionate to the temperature. An actual tachycardia with a pulse rate of 150 to 160 per minute may be encountered in the more severe types of diphtheria. When antitoxin is given early and in sufficient dosage a prompt improvement in the pulse rate is noted. The heart, however, must be watched for several weeks after every case of diph-

theria and the child kept at absolute rest so long as there is any abnormality in the pulse. A systolic murmur, indicating a relative insufficiency of the mitral valve from dilatation is often observed after diphtheria and may persist for weeks. (Irregularity of the pulse is also common during convalescence.) The chief danger, however, lies in the development of heart block. This usually occurs at the beginning of the second week in cases which did not receive antitoxin early. There is a sudden drop in the pulse-rate which may fall as low as 30 to 40 per minute; there is pallor, apathy and prostration and the child often vomits and complains of epigastric pains. These symptoms are the most serious ones that can be encountered in diphtheria, aside from paralysis of the muscles of respiration which is a later development. The majority of cases of complete heart block terminate fatally within a few days.

Diphtheritic paralysis occurs more frequently in adults and older children than in young children; it is seldom seen in infants. Cases which have received a sufficient dosage of antitoxin during the first twenty-four hours of the disease rarely, if ever, develop paralysis.

The symptoms are those of a multiple neuritis. In the majority of cases symptoms do not occur until the second or third week. Paralysis of the soft palate is the first symptom noticed, manifesting itself by nasal voice, regurgitation of food through the nares, and difficulty in swallowing.

The eye-muscles are frequently affected early, and loss of accommodation, strabismus and ptosis are the disturbances encountered here. When the extremities take part in the paralysis the patient complains of muscular weakness, with tingling and numbness, gradually increasing in severity until he is perhaps unable to walk or use his arms, although complete paralysis is rare. The distribution of the paralysis is symmetrical. Sensation is markedly impaired and the knee-jerk lost, even, at times, without the existence of paralysis. The prognosis as to ultimate recovery is good, although the course is

variable, some cases continuing for several months before improvement sets in. Death may result from paralysis of the respiratory muscles or from involvement of the vagus. This is characterized by vomiting, epigastric pains and weak, irregular pulse.

Extension of the membrane to the nose is indicated by nasal obstruction with an acrid, offensive, muco-purulent discharge and increased swelling of the lymphatic glands at the angle of the jaw, together with involvement of the submaxillary glands. Epistaxis occurring during diphtheria is always a suspicious symptom. Owing to the large absorbing surface brought in contact with the toxin, constitutional symptoms are markedly aggravated, and prostration becomes extreme. *Primary nasal diphtheria* is, as a rule, not nearly as grave a condition as the secondary form, although such a case may infect another child with a faucial diphtheria of the usual severity.

Extension into the larynx is indicated by progressively increasing dyspnea, cyanosis, and a croupy cough. The process may result in complete stenosis of the larynx, with death from suffocation.

Septic diphtheria is a term applied to a severe type of the disease with pronounced throat symptoms, foul breath, marked adenopathy and high fever.

Primary Laryngeal Diphtheria.—*Laryngeal diphtheria* or *membranous croup* is a primary infection of the larynx characterized by the formation of a false membrane (croupous exudate) upon the laryngeal mucous membrane. The false membrane may remain confined to the larynx, or extend down into the trachea. Often it is accompanied by a scanty tonsillar exudation. Laryngeal diphtheria differs clinically from faucial diphtheria in the absence of the marked constitutional symptoms and adenopathy observed in the former. This may be due in part to a difference in the type of organism showing a predilection for the larynx rather than for the throat. A better explanation, perhaps, is the fact that the mucous membrane

of the larynx does not absorb toxin as readily as that of the fauces.

The onset is insidious, with moderate fever, croupy cough, and hoarseness. During the first twenty-four hours the symptoms are laryngeal; the child is hoarse and has a hard, croupy, non-productive cough. This may at first be mistaken for catarrhal croup but the cough and difficulty in breathing persist throughout the day and progressively increase. Complete aphonia soon develops. Recession of the epigastric region during inspiration indicates an on-coming stenosis of the larynx and with the increasing stenosis breathing becomes a purely voluntary effort. The child now sits erect, and with every effort at inspiration the accessory respiratory muscles are thrown into action. The body surface is cold and cyanotic, and the child becomes drowsy and later comatose, dying from asphyxia. Death may result in a few days from the time of onset, unless antitoxin has been used early. As soon as labored breathing develops intubation should be performed.

Pseudo-Diphtheria differs from true bacillary diphtheria both etiologically and symptomatically, being caused by a streptococcic infection of the throat. It may develop independently or complicate scarlatina, measles, etc. As a complication of scarlatina it appears, however, more frequently and more virulently than in any other form.

The *clinical course* of pseudo-diphtheria differs distinctly from that of bacillary diphtheria. In pseudo-diphtheria there is pronounced inflammation of the pharynx and tonsils with redness, swelling and pain. It begins abruptly, with high fever, lassitude and headache. Small, white or yellowish patches develop upon the tonsils, they become darker in color and may coalesce, but seldom spread beyond the tonsils, in this respect differing distinctly from the membrane of true diphtheria. The membrane is more friable than that of diphtheria, and can usually be detached without difficulty and without injury to the under-lying mucous membrane.

Marked swelling of the lymphatics seldom takes place. It may occur in epidemic form as the so-called "septic sore throat." The duration is from four to five days and although constitutional symptoms are severe during the height of the disease, the throat symptoms being particularly distressing, still they are never of a dangerous character and sequelæ are rare. Paralysis never follows pseudo-diphtheria, nor is extension to the larynx to be feared.

Prognosis.—The prognosis of diphtheria depends so definitely upon the question of early treatment with antitoxin that other factors are only of secondary importance. Unfortunately many cases are not recognized in time to receive prompt treatment or the administration of antitoxin is delayed. Aside from the question of treatment the following points should also be considered:

The age is of importance, as diphtheria is uniformly more fatal in infants than in older children. Adults present the best chances, but they are more subject to paralytic sequelæ.

The character of the epidemic is of some importance, as there is a difference in the virulence of cases from time to time. A most virulent diphtheria, however, may originate from an apparently mild diphtheritic sore throat, and *vice versa*.

The appearance and distribution of the membrane offer suggestions for a prognosis, but here again errors are liable to occur. Cases with extensive membranous deposit may make a good recovery without any sequelæ while one with a scanty membrane may be accompanied by grave toxemia.

The time at which treatment was begun and the patient's general condition, therefore, offer the safest guides in determining his chances for recovery. So long as the pulse remains good and the child can take nourishment satisfactorily the case should not be despaired of.

In *membranous croup* the prognosis is more favorable than in secondary laryngeal diphtheria.

During convalescence there is danger of cardiac failure from

neuro-muscular deficiency. This may appear as progressively increasing heart weakness with irregular pulse or occur suddenly or after some physical exertion. The child is seized with epigastric pain and vomiting; there is dyspnea; cyanosis; small, irregular pulse and collapse. Heart block often develops in these cases. If the first attack does not prove fatal there is usually a recurrence with a fatal issue.

Bronchopneumonia occurring with diphtheria is very unfavorable; it is especially serious when complicating croup.

Diagnosis.—The throat of every child that is acutely ill should be routinely examined no matter whether it complains of sore throat or not. In the event of an exudate or membranous deposit being discovered on the tonsils a culture should be made for determining the presence of the Klebs-Loeffler bacillus. When the clinical indications are strongly in favor of the diagnosis of diphtheria, antitoxin should be administered without waiting for the results of the culture.

The differential diagnosis rests mainly between *pseudo-diphtheria* and *follicular tonsillitis*. *Pseudo-diphtheria* is abrupt in onset; lymphatic swelling is absent in primary cases, fever is high, and the throat is markedly reddened and swollen, and there is considerable pain on swallowing; the exudate is purely fibrinous, and it does not tend to spread beyond the tonsils. Secondary cases occur during the febrile period of scarlet fever. Paralysis never follows, and although septic symptoms may be present the specific toxic symptoms of diphtheria are absent. The membrane is thinner, can be removed without bleeding, and is usually of a yellowish color, later becoming dirty.

In *follicular tonsillitis* both tonsils are uniformly swollen and covered with small, round, white spots, which are not adherent to the mucous membrane, but consist of plugs of exudation filling the crypts of the tonsils, from which they can be readily wiped off.

Treatment.—A child with a sore throat should be isolated

and if the case proves to be one of diphtheria this isolation must be continued until recovery is complete and two successive negative cultures from the throat have been obtained. Isolation and sick-room hygiene are carried out as in the case of other contagious diseases, like scarlet fever, for example. The dishes used by the patient should not leave the sick-room while all articles of clothing and laundry should be disinfected before they are taken out of quarantine. The child should use rags or paper handkerchiefs and these should be burned.

Other members of the family who have been exposed to the patient should receive an immunizing dose of antitoxin unless they present a natural immunity to the disease.

The Schick Test. Schick (*Muenchner Med. Wochenschrift*, 1913) devised a practical means of determining individual susceptibility to diphtheria which has been used for the purpose of determining whether or not children exposed to diphtheria should receive an immunizing dose of antitoxin. The reaction depends upon the observation that certain individuals possess natural antitoxin in their blood while others do not, and that when a small dose of diphtheria toxin is injected intradermally into an individual not possessing a natural immunity a marked local reaction results. In the case of individuals who are immune there is no reaction. A positive reaction therefore indicates that the person is susceptible to diphtheria and is in danger of becoming infected.

The test is performed by injecting $\frac{1}{50}$ of the minimum lethal dose of diphtheria toxin for a guinea pig, diluted with 0.1 c.c. salt solution, intradermally into the skin of the forearm. A similar injection of plain salt solution is used as a control. A positive reaction is indicated by the appearance, within 24 or 48 hours, of an area of redness which is at its height on the third day and then gradually fades, leaving a brownish discoloration and slight itching and desquamation.

The *diet* should be concentrated and highly nutritious, and stimulation is of the greatest importance as soon as the toxic

influence of the diphtheria virus upon the heart and nervous system becomes apparent. A teaspoonful of whisky well diluted with water or milk, and administered every three hours, suffices for the average case; but where there is prostration and failing heart the quantity must be increased accordingly. Absolute rest is to be enjoined during convalescence as well as during the disease in all cases showing cardiac weakness, in order to avert a possible sudden death from acute dilatation of the heart.

In regard to *local treatment*, it can be stated that all measures which in any way give the patient pain or discomfort and require physical restraint, will do more harm than good. A gargle of *permanganate of potash* (1 to 1,000) is useful. Alcohol diluted with four or five parts of water is also a good gargle and is more agreeable to the patient than the permanganate.

In *nasal diphtheria* the nasal chambers should be kept as free from secretion as possible. A douche of warm normal saline solution should be used about three times daily, great care, however, being exercised to avoid getting the fluid into the Eustachian tubes.

In *laryngeal diphtheria* a steam spray or a croup kettle adds to the comfort of the case. Intubation or tracheotomy should not be put off until the child becomes exhausted from its dyspnea. Intubation is the operation of choice.

In all forms of diphtheria, but especially in croup, it is well to keep the air of the room moist and at a temperature of about 70° F., if this be practicable. Good ventilation of the sick-room must of course be carried out. The placing of disinfectants and antiseptics about the room is of no value.

Serum Therapy.—Shortly following the discovery of diphtheria antitoxin the mortality of diphtheria was reduced from over 50 per cent to about 12 per cent (Babinsky) and the mortality of laryngeal diphtheria from 73 per cent to 27 per cent (*The American Pediatric Society Report*, 1897). In recent years, through the earlier administration of antitoxin

and the use of larger doses the mortality has been still further reduced. Antitoxin may be looked upon safely as a sure cure for diphtheria and "if carelessness, ignorance and inertia were excluded, the death-rate to-day could be zero" (Place, *The Oxford Medicine*, 1921).

Antitoxin, as the name implies, is an antidote to the toxin generated by the diphtheria bacillus but it does not destroy the bacillus. In order to get the best effect from antitoxin, therefore, it must be administered early in order to prevent the toxin in the blood from exerting its specific effect upon the tissues. If the disease has not been promptly arrested, as shown by the continuance of the fever and the persistence of or spread of the membrane a second dose must be administered. A large initial dose in the majority of instances arrests the progress of the disease, making a second dose unnecessary and is therefore to be advocated.

The *dosage* of antitoxin is purely arbitrary and since no more harm can come from a large dose than from a small one it is better to err on the safe side and give the comparatively large dose. The present good commercial brands of antitoxin are concentrated and the amount to be injected has therefore been reduced considerably; also, most of the serum has been removed and for this reason serum rashes are rarely encountered. From personal experience I would recommend the following doses:

Mild cases, with purely tonsillar exudate, 5000 units. *Moderately severe cases* with extension to the pillars or uvula, 10,000 units; *severe cases* with *nasal involvement*, 20,000 units given intravenously. *Laryngeal cases*, 10,000 units repeated in from six to twelve hours if necessary.

The injection may be made subcutaneously or intramuscularly; the latter method is preferable. The best site for a subcutaneous injection is the lateral chest region below the axilla. Intramuscular injections may be made into the outer side of the thigh or into the buttocks. A prompt drop in the temperature and pulse rate together with a decrease in the faucial

hyperemia is noted when sufficient antitoxin has been administered. The membrane ceases to spread and on the following day it shows signs of loosening from the mucous membrane. If these signs of improvement do not occur, a second and larger dose (an additional 5000 units) should be administered.

Homeopathic remedies are of value both for the local and general disturbances caused by the diphtheritic toxin. The *red iodide of mercury* is especially useful for the throat symptoms; *belladonna* is indicated by the fever and inflamed throat and should be alternated with the *mercury*. In the advent of nephritis, the *bichlorid of mercury* is indicated. *Apis* is indicated when there is much edema of the fauces. When toxemia is present *arsenicum* and *rhus tox.* are indicated.

Cardiac weakness calls for stimulation; whiskey and *caffeine sodiobenzoate*, 1 to 2 grs. hypodermically. When there is vomiting and signs of rapidly progressing heart failure *morphia* hypodermically is the best remedy. Nothing should be given by mouth and enteroclysis may be employed.

Post-diphtheritic paralysis.—*Gelsemium*, *causticum* and *phosphorus* are the most useful remedies. *Strychnia* is extensively used but without distinct benefit. The indications for *gelsemium* are paralytic weakness of the extremities, paralysis of the eye muscles and cardiac irregularity. *Causticum* and *phosphorus* present symptoms of definite nerve and cord lesions.

GLANDULAR FEVER.

The term "glandular fever" is used to describe a peculiar type of acute infection in which enlargement of the superficial lymph-nodes and fever are the chief clinical manifestations. The condition was first described by Pfeiffer in 1889 and was considered by him to be a specific acute infectious disease. He did not, however, succeed in isolating a specific organism and while epidemics from various sources have been reported, still glandular fever is not universally recognized as a specific infectious disease.

In the epidemics which have been reported, several cases in a household have usually been observed. It is most frequently seen between the ages of two and eight years.

Symptoms.—The onset is abrupt with symptoms suggestive of an upper respiratory infection, namely, fever, slight redness of the throat with discomfort and at times the child complains of a painful stiffness of the neck muscles. This symptom is probably due to the associated adenitis. Swelling of the cervical glands is the most marked clinical manifestation, the posterior cervical chain being chiefly involved. The swelling is so pronounced that the glands can be seen as well as palpated. They are painful to the touch but there is no redness of the overlying skin and suppuration does not set in. Associated with the cervical adenitis there is more or less general involvement of the superficial lymph nodes so that enlarged glands in the axilla and groins can usually be demonstrated. Enlargement of the spleen has also been frequently noted as well as enlargement of the liver. In some of the more severe cases a transient albuminuria has been noted.

The duration is from a week to ten days, depending upon the severity of the infection. No fatalities or sequelæ have been reported. With the subsidence of the fever the glands gradually return to normal and abscess formation is not to be anticipated.

The chief interest to be attached to glandular fever is the question of *diagnosis*. We can only accept the diagnosis of glandular fever in a case of acute febrile generalized adenitis in which an acute throat infection such as a streptococcus sore throat can be ruled out. Streptococci may, however, set up an infection of the cervical glands without leaving much evidence of their activity in the mucous membrane of the throat. Often the case is not seen until two or three days after the initial symptoms developed and the pharyngitis or tonsillitis which was present at that time has already subsided. The infection, however, is still active in the lymph-nodes which

accounts for the persisting fever and glandular swelling. Acute cervical adenitis with high, irregular fever which may last for several weeks is not an uncommon condition and in these cases the throat may appear to be negative unless the child was seen at the time of onset of the infection or unless a more than cursory examination of the tonsils and posterior nares is made. Fortunately these cases usually clear up without suppuration as in the case of glandular fever but the temperature is more of the septic type and of longer duration.

The *treatment* of glandular fever is that of any other mild infectious disease. In the early stage the instillation of a ten per cent aqueous solution of argyrol into the nose may be of advantage. *Belladonna* 2x is useful at this stage for the fever, throat discomfort and neck symptoms. As the glands become swollen and sensitive *mercurius solubilis* 3x trituration may be given in alternation with the *belladonna*. Local applications to the glands are unnecessary.

TYPHOID FEVER.

Typhoid fever is a self limiting disease, caused by the bacillus of Eberth. The bacillus is found in large numbers in the discharges from the bowels of a patient suffering from typhoid fever. It can also be recovered from the spleen, liver, Peyer's patches and the mesenteric glands. During the first week of the disease, positive blood cultures may be obtained in practically 100 per cent of all cases. The organism is, however, gradually destroyed in the blood stream and during the second week the percentage of positive blood cultures falls to about 70 per cent and in the third week to about 30 per cent. It can rarely be recovered from the blood during convalescence, but at this time it may be still cultured from the urine and feces. In fact, in some patients the bacilli persist in the urine and feces for a long time after convalescence and such an individual consequently becomes a "*typhoid carrier*." It is also believed that

the gall bladder may harbor the typhoid bacillus for a long time.

The anatomical lesions are inflammation of Peyer's patches and of the solitary follicles in the ileo-cecal region with tendency to ulceration and enlargement of the spleen. A maculopapular eruption of rose-colored spots appearing mainly upon the abdomen is one of the pathognomonic signs of typhoid fever, but like ulcerative lesions of the intestines it is not so constantly associated with the disease in children as in adults. The typhoid bacilli have been demonstrated in these spots. The accompanying symptoms are fever of a characteristic type; prostration and disturbances in the nervous system; more or less diarrhea and wasting. Here again it is not as typical as in adults. The fever is more irregular, and the duration is shorter, as a rule. On account of the absence of pronounced ulceration of the bowel in the second week, the temperature does not show the septic course assumed in adults at this time. This condition, however, is not to be absolutely excluded. The associated symptoms are usually milder and diarrhea may not appear until in the later stages of the disease. There is, however, a severe type of typhoid fever occurring in children that may present every unfavorable phase of the disease as it is encountered in the adult, not barring copious hemorrhages and perforation of the bowels, but as a rule the gravity of these cases depends more upon the degree of toxemia than upon anatomical lesions.

Only in recent years has the fact that typhoid fever is common during childhood been recognized. Many mild cases were looked upon as a simple continued fever, while more pronounced ones received the appellation infantile remittent fever, or they were diagnosed worm fever. Some confusion as to the gravity of the disease is still to be detected in the writings upon this subject. My own experience leads me to the conclusion that we encounter both mild and grave cases of typhoid fever in children just as in adults, with the exception however that

the mild cases predominate. Aside from this I can see very little difference between the disease as it occurs in childhood and in adult life. In infancy typhoid fever is rare and runs a grave course. In childhood, however, we seem to have more vitality and recuperative power than in adult life and the child stands a better chance of pulling through than an older individual.

Etiology.—Infection takes place through the alimentary tract. The commonest source of infection is drinking water that has been contaminated with the dejecta of typhoid fever patients.

Milk is a common carrier of the infection. As the bacilli grow rapidly in milk, the adulteration of this commodity with contaminated water becomes a grave matter. A frequent cause of milk contamination is brought about by the rinsing of milk cans with polluted water or the handling of the milk by "typhoid carriers." The possibility of the germ entering the system through the inspired air is doubtful. Contagion is not uncommon as shown by the frequency of the disease among nurses. The house fly is also most likely a potent factor in the spread of the disease.

Typhoid fever is rarely encountered before the second year, but there is no doubt that it does occur during infancy. A number of authentic cases are on record, and I have personally encountered it. In two reported epidemics of wide distribution 1 per cent of the cases occurred in infants under two years old. The mortality is high in the infantile cases.

The majority of cases are seen after the sixth year. Boys are more frequently attacked than girls. Epidemics are more prevalent in the fall than at other seasons. Modern sanitation has practically eliminated typhoid fever from most of our larger cities.

Pathology—The pathologic lesions are not as marked as in the adult, and the usual explanation given for this difference in pathologic findings is the fact that the child's gut is still

anatomically immature. The first change observed in the intestines is a catarrhal inflammation of the lower portion of the ileum, together with swelling of the solitary follicles and Peyer's patches in the ileo-cecal region. The cecum and colon are moderately involved in the catarrhal inflammation.

As the process continues round-cell infiltration into the lymphoid structure constituting the swollen follicles and patches takes place, with the formation of elevated plaques and shot-like projections. The amount of infiltration, however, seldom attains to the degree observed in the adult, and, instead of necrosis from compression of the blood-vessels supplying the affected area setting in, it usually terminates by fatty degeneration and resorption of the infiltration. For this reason the course is shorter and more benign, and ulceration of the bowels is much rarer than in adults. In older children, however, the same lesions are to be found that characterize typhoid fever in adults. With the breaking down of the infiltrated areas, deep oval ulcers, their long axis corresponding to the direction of the bowel, are found. Smaller, irregularly-scattered ulcers result with the breaking down of the solitary follicles. The slough is more frequently superficial separating without the production of a deep ulcer and unattended by the septic fever observed in adults at this stage. Grave symptoms are more frequently dependent upon toxemia than upon anatomical lesions. General infections without localization is also possible. According to Mallory these pathologic lesions result from a specific action of the typho-toxin upon the endothelial cells of the blood-vessels, most marked at the site of the infection (intestinal submucosa). Cell proliferation results, these cells acting as phagocytes and many of them fuse and become giant cells. A secondary result is clogging of the capillaries with these cell clumps and in this way necrosis of the tissue results.

The changes found in other organs are swelling of the mesenteric glands, swelling of the spleen, which is soft and pulpy; parenchymatous degeneration of the heart, liver and kidneys.

Hypostatic pneumonia, bronchitis of the finer tubes and bronchopneumonia are commonly associated with typhoid fever. These lesions are usually due to a secondary infection, although the typhoid bacillus may incite any of these complications and even act as a pus producer, notably when it invades the joints or medullary canal. Slight pathological changes in the kidneys are common and severe lesions may occur. Bacilli are present in the urine in about 20 per cent of cases during the third and fourth week (Park), and the urine may become cloudy from their presence.

Symptoms.—The onset of typhoid fever is gradual in the majority of cases being preceded for a day or two by prodromal manifestation, such as general malaise; headache; restless and dream-disturbed sleep; anorexia and constipation. There may be slight chilliness recurring for several days, but rarely a decided initial chill. Nosebleed is less frequent than in adults, but abdominal pain is more common. The temperature now begins to rise in a characteristic manner. Morning remissions are marked, but the fever rapidly reaches its acme, usually in from four to five days; in adults this is not attained until the end of the first week, and there is a more gradual step-like rise in the temperature.

At times the temperature rises abruptly instead of ascending gradually. This is more common in children than in adults. The temperature soon reaches its maximum evening rise (103 deg. to 104 deg. F.) and by the end of the second week a rapid decline in the temperature is the rule in such cases. On the other hand, an abrupt beginning with high fever (105 deg.) and early delirium is characteristic of the gravest (fulminating) form of the disease, namely, acute typhoid septicemia. After the acme has been attained the fever presents a continuous remitting type. The remission occurs in the morning, and the exacerbation in the evening; in severe forms, with high temperature, the remissions are not as marked as in milder cases. Toward the end of the second week (about the twelfth

day) the morning remission becomes more pronounced, and soon a lowering in the evening rise is noticed. The temperature now falls by lysis, and in the course of from a few days to a week the stage of defervescence is completed. Accordingly, a typical, uncomplicated case of moderate severity occurring in a child under ten years old pursues a course of from fifteen to nineteen days. Severe cases, or such in which complications occur, run a much longer course or prove fatal.

The age of the child apparently exerts an influence upon the duration of the fever. In children of five years the average duration is 15.7 days; at eight years, 18.3 days, and at ten years 20.3 days (Montmollin). Cases of short duration however are very likely to have reinfections and so in the end most typhoid fever cases run their allotted three to four weeks before full immunity is established.

The symptoms occurring during the first stage are fever, accompanied by prostration, gastric derangement and marked indifference. The face is pale, and the cheeks usually flushed. The tongue is heavily coated, the lips dry and the breath offensive. During the second and third week the prostration and indifference increase excepting when the patient becomes actively delirious, the lips are cracked, often bleeding; there is pallor and a characteristic waxy appearance of the extremities develops as a result of the poor peripheral circulation.

The temperature is not always an index of the severity of the infection. While abrupt onset with early hyperpyrexia indicates an intense infection, still a weak heart and low vitality may be the reason for failure to react against the disease and consequently we may have most serious cases with moderately high fever. Cases are on record in which no rise of temperature occurred, and still grave symptoms were present. In such the prognosis is bad. A sudden rise of temperature during the course of the fever usually indicates a complication, while a sudden fall means hemorrhage or perforation.

The tongue is heavily coated with a light-yellowish fur.

This coating wears off in places, exposing the slightly swollen papillæ as red specks. A red streak down the centre is likewise produced by the tongue rubbing against the upper central incisors during its propulsion. Only in severe and protracted cases does the tongue become brown and cracked.

Cracking and bleeding of the lips is common in children because they constantly pick at the same unless restrained.

The bowels are usually constipated in the beginning, but they may become loose as the fever progresses. In cases marked by severe bowel symptoms the stools are thin and watery; often involuntary. The abdomen is prominent, and tenderness and gurgling are found in the right iliac fossa upon pressure. This is due to the accumulation of fluid in the lower ileum and may be present even when there is constipation. Gas forms plentifully, but owing to the parietic condition of the gut it is not readily expelled. The typical typhoid stool is loose and of a dirty, yellow color, being appropriately described as the "pea-soup stool." It has a characteristic penetrating offensive odor, which may cling stubbornly to the patient. During the second week, when delirium sets in, the stools are often involuntary; this condition may remain to the end in adynamic cases.

The eruption is not as constant and is less abundant in young children than in adults; it is found upon the abdomen and lower portion of the chest, developing in crops. It is absent in perhaps 20 per cent of cases (Jacobi). The first crop appears about the eighth day, successive crops appearing for a week or longer. The spots consist of small, rose-colored macules, disappearing on pressure. They may spread to the neck and lower extremities, and in serious cases with septic infection petechiæ may develop.

The spleen becomes enlarged early in the disease; in fact by the end of the first week it can usually be felt at the border of the ribs. It may serve as an index to the progress of the disease, its return to normal size during the middle of the third week auguring a good prognosis. If it fails to diminish in size there will be a relapse (Jacobi).

The pulse furnishes valuable data for diagnosis early in the disease. There is a characteristic slowness about the pulse of typhoid fever in the first week which is not, however, as marked in children as in adults. Nevertheless, the pulse does not increase in rate to the same degree that it would be found in most other acute febrile conditions. With the progress of the fever, however, it becomes rapid and feeble. We should, therefore, always suspect enteric fever whenever a febrile condition is encountered in children in association with a relatively slow pulse-rate in the early stage. The opposite condition holds good in meningitis. The dicrotic pulse, so characteristic in adults, is observed only in older children.

The disturbances of the nervous system are apathy, prostration and cerebral irritability. The child is often exceedingly cross and slow in answering questions and obeying requests. Delirium is usually present, especially during the night, and if the child is particularly susceptible to the typhoid poison, symptoms resembling meningitis may develop. Thus, dilated pupils, retraction of the head, twitching of the muscles of the face and extremities, crying out in sleep and stupor are frequently encountered. They disappear with the fall in the temperature. Cases that are very toxic may have either an active, excitable delirium or a low, muttering delirium, and twitching of the tendons, gritting the teeth and crying out in sleep, together with complete loss of control over the bladder and bowels will be noted. A true meningitis may complicate typhoid fever in rare instances.

The urine may become albuminous from acute parenchymatous degeneration of the kidneys ("nephro-typhus"). The bacillus is usually present in the urine in such cases, which accounts for the albuminuria. Actual nephritis is rare. During convalescence the urine is usually increased although it is possible to incite a polyuria during the course of the fever by making the patient drink copiously of water.

The blood undergoes no important changes in the early

stages of the disease; by the third week a decided anemia has developed, due to a reduction both in the number of red corpuscles and in the amount of hemoglobin. A moderate leucopenia with increase of the large mononuclear leucocytes is characteristic. Leucocytosis does not appear unless perforation or secondary infection occurs.

Besides the rose-spots, sudamina frequently develop upon the skin, mainly on the chest and abdomen. They appear in the later stages of the disease. At this period profuse and debilitating sweats may occur, with subnormal temperature. A subnormal temperature during convalescence is the rule.

Bed-sores, boils, phlebitis and abscesses in various regions are seen in septic cases and in the debilitated.

Abortive Type.—Instead of running its full course typhoid fever may abort at any stage. The child may be taken ill with such characteristic symptoms as nosebleed; ascending fever; iliac tenderness; dry, coated tongue and rose-spots, but by the tenth or twelfth day the temperature has reached normal. A positive Widal reaction verifies the diagnosis in these cases.

Reinfection and Relapses; Recurrences.—Reinfection may be said to occur in about 10 per cent of cases. It usually occurs during the first two weeks of convalescence, but a sudden rise in the temperature and a return to the original fever curve, together with the reappearance of symptoms, may set in during the latter part of the third week before the evening temperature has yet become normal. A relapse indicates a reinfection with germs that have escaped destruction and it is accompanied by the symptoms of the original attack. A fresh crop of rose-spots usually appears. The average duration is from ten to fourteen days. The symptoms are usually mild, but death may occur during a relapse. Relapses are common in cases that have run a mild or short course and have not developed full immunity. One attack confers immunity for a life time and second attacks of typhoid fever are exceedingly rare. Usually a so-called second attack is one of paratyphoid fever.

Among the complications, bronchitis and bronchopneumonia are the most frequent. Bronchitis is almost a constant accompaniment of typhoid fever. Bronchopneumonia is not uncommon; this complication is a frequent immediate cause of death in the grave types of the disease. A lobar pneumonia may also complicate typhoid fever. This may be due to a mixed infection with the pneumococcus or it may be due primarily to the typhoid bacillus. Otitis media; bedsores; circumscribed suppurative processes; phlebitis and intestinal hemorrhages are occasionally seen. Fatal hemorrhages and perforation are rare, but perhaps not as rare as is generally supposed. Abscess of the lung, empyema and septic parotitis are also among the rare complications, usually seen only in hospital practice. They are almost always fatal.

Hemorrhage occurs most frequently during the third week. Its indications are collapse and a rapid fall of the temperature. Death may occur before blood is expelled. The coagula may be felt in the ileo-cecal region. Perforation is less common than hemorrhage and presents the most serious of all accidents. Characteristically it is preceded by sharp abdominal pain followed by collapse, and usually intestinal hemorrhage. The condition, however, may be masked and not suspected until peritonitis develops.

Other conditions which have been found associated are ulceration of the mouth, throat and genitals; peritonitis; suppurative synovitis and osteitis; nephritis; tuberculosis. Endocarditis is rare but myocardial degeneration is frequently found in the severe types of infection.

Sequelæ affecting the nervous system are transitory aphasia; multiple neuritis; chorea and insanity, all fortunately rare.

Prognosis.—The prognosis is, on the whole, more favorable in children than in adults. Perhaps the chief reason for this is the average shorter duration of the fever, the greater tolerance on the part of the heart and the lesser liability of severe hemorrhage and intestinal perforation, but we must bear in

mind that the previous health of the child and the development of one of the graver complications must be carefully considered in estimating the prognosis. In young infants the prognosis is grave. The mortality rate is not very uniform, thus Holt has placed it at 5.4 per cent; Steffen at 6.7 per cent; Henoch at 7.5 per cent and Baginsky at 9 per cent.

The age is an important factor; the intermediate ages are the most favorable. The pulse and temperature are ordinarily a safe guide, but as stated above the height of the fever does not always indicate the degree of infection. It is, therefore, best to go direct to the heart, auscultating daily to ascertain the condition of the heart muscle. When the pulse-rate remains relatively low in comparison with the fever and its volume is good there is no immediate danger to be feared. A rapid pulse, especially when this occurs early in the disease, is an unfavorable omen.

Regarding the temperature, the absolute height of the fever in uncomplicated cases is of prime prognostic importance. "With every day that the temperature retains its high range without interruption the danger to the patient grows" (Klemperer). We can usually judge of the course that the fever is about to run after we have observed the case up to the end of the first week. It is rare for the temperature to rise above the point attained at this time. The duration of the fever in cases of abrupt onset with high fever is, as a rule, short. This does not, however, apply to fulminating typhoid. The daily variations in the fever are also important prognostic points; the greater the daily remissions, the less destructive to the organism will be the fever, while a continuously high fever with but slight diurnal variation, and one that is not influenced by baths, etc., offers an unfavorable prognosis.

Complications, such as pneumonia, septic infection, hemorrhage and tympanitis always render the prognosis more unfavorable. In the fatal cases coming under my notice there was present, as a rule, a grave secondary infectious condition, such

as septic parotitis, empyema, pulmonary abscess and osteomyelitis. Acute typhoid septicemia is also fatal in the majority of instances. Geohegan reports a fatal case from perforation and hemorrhage in a child under two and a half years old.

Diagnosis.—Aside from the pathognomonic symptoms of typhoid fever, viz., continued fever of a definite type, rose-colored spots, tympanitis with gurgling and tenderness in the right iliac fossa, enlarged spleen and pea-soup stools, there is at our command the blood test of Widal and the urinary test (dialo-reaction) of Ehrlich. Unfortunately for the general practitioner, the former is difficult to carry out, requiring special laboratory facilities and expert technique in bacteriology. In every large city, however, there are pathological laboratories where this test can be made so that it is rarely necessary for the physician to be especially equipped. Widal's test consists of the introduction of a few drops of blood from a patient suffering with typhoid fever into a pure culture of typhoid bacilli. A microscopical examination reveals a prompt formation of clumps consisting of the agglutinated bacilli, which have also lost their motility. The reaction is one of infection and immunity, indicating that a toxic substance has been formed in the blood serum, which is capable of destroying the motility of the germs causing the disease, and also inducing their agglutination. Dried blood drops, collected on unglazed paper, may be used for the test. In dilutions of 1 to 40 or 50 with fresh bouillon cultures of typhoid bacilli the characteristic reaction may be obtained. The Widal reaction may be observed on the fourth day of the disease, but it is usually delayed until the eighth day. It continues throughout the fever and may persist for some time after the recovery.

The frequently-recorded negative results should not weigh heavily against this most valuable diagnostic adjuvant, as faulty technique is probably more to be blamed than the test itself. The proportion of cases in which a definite reaction occurs and the time of its appearance, based on an extended Health Depart-

ment Laboratory experience, is given by Park as follows: 20 per cent gave positive results the first week, 60 per cent in the second week, 80 per cent in the third week, 90 per cent in the fourth week. In 88 per cent of the cases in which repeated examinations were made (hospital cases) the reaction was found at some time during the fever. Its late appearance, usually not before the eighth day, renders it less valuable than blood cultures as an early sign.

The diazo-reaction is a valuable corroborative test, but it is also obtained in acute miliary tuberculosis and in rapidly progressing pulmonary tuberculosis. In fact, a large number of infectious conditions will give this reaction, notably measles.

Cases without intestinal localization will present difficulties in diagnosis. In such, only a bacteriological examination of the blood will solve the problem. Many of the acute typhoid septicemias are of this character. They present the picture of a profound toxemia with high fever and early delirium. Death may occur before it is possible to reach a diagnosis, and the post-mortem findings may be entirely negative (Osler).

From *malarial fever* it can be differentiated by means of a blood examination to ascertain the presence or absence of the malarial parasite, and by the temperature curve.

Meningitis.—A strong point of difference between meningitis and typhoid fever is the behavior of the pulse. In typhoid fever it is relatively slow in the beginning, becoming rapid toward the end of the disease; in meningitis the pulse rises proportionately with the fever in the beginning, but becomes slow and irregular towards the close of the case. Furthermore, in meningitis the abdomen is retracted, the bowels are constipated throughout, and paralyses of the cranial nerves are to be observed. The reflexes are exaggerated, and Kernig's sign may be elicited. None of these symptoms are present in typhoid fever. Contraction of the flexors of the legs may develop in protracted cases of typhoid fever as a result of nutritional disturbances of the muscles or possibly a neuritis

but this should not be confused with the Kernig sign. Meningeal irritation is common, but true meningitis is very uncommon. In typhoid fever of the cerebro-spinal type, it may be necessary to resort to lumbar puncture before a positive diagnosis can be made.

Acute miliary tuberculosis may present difficulties in differential diagnosis. Aside from the absence of the Widal reaction in tuberculosis there is a more rapid pulse and greater irregularity in the course of the fever. Often the "inverted type" of fever is noted. The spleen is less likely to be enlarged than in typhoid fever. The most characteristic sign of miliary tuberculosis, however, is rapid respirations and cyanosis, more or less pronounced. An old tuberculous lesion may be demonstrable. Meningitis is commonly associated. The most difficult cases to differentiate are those in which abdominal symptoms are the predominating feature.

Early pronounced localization of the infection in some other system than the intestinal tract may lead to confusion. As Osler emphasizes, the brunt of a very acute infection may fall upon the cerebro-spinal, the pulmonary, or the renal system. Typhoid lesions in the appendix may lead to a suspicion of appendicitis. The fact, however, that malaise and fever have preceded the development of the iliac tenderness should throw out the diagnosis of appendicitis which begins with general abdominal pains followed by vomiting, then appendicular tenderness, rigidity of the right rectus muscle and lastly fever and rapid pulse.

Treatment.—The patient should be put to bed in a room that can be thoroughly ventilated, and one from which all unnecessary furniture and draperies have been removed. Provision must be made for the disinfection of the stools and urine, which can be accomplished by the use of an active germicide. A strong solution of chloride of lime, Platt's chlorides, or carbolic acid (5 per cent solution) is to be poured over the stools as soon as they are passed, and allowed to act upon them for

several hours before they are emptied into the water-closet. All towels, napkins and sheets soiled by the patient should be boiled in order to render them sterile.

The diet is of the greatest importance. Owing to the intestinal lesions, solid food must be withheld until at least a week after disappearance of the fever, diarrhea and abdominal tenderness. Where abdominal symptoms have been pronounced during the fever, it is better to wait even longer before resuming solid food. In the milder class of cases we may return to semi-solid food on the fifth day after the temperature has ceased to rise above 100 deg. F., gradually returning to solid food. Such articles of diet as thoroughly cooked cereals; poached eggs; milk toast; the soft portion of a baked apple; baked potato, etc., should be selected at this time.

Of late there has been a decided reaction against the strict dieting of the typhoid fever patient and semi-solid foods are prescribed by many clinicians during the height of the fever. While this is permissible in adults especially when the patient is hungry and not delirious I find that children do much better on a restricted diet. The chief foodstuff which they require to maintain nutrition and avert dangerous destructive tissue changes is carbohydrate and this can be added to the milk and thus given in liquid form. By this means we also supply a large amount of water which is one of the most important agents in the treatment of the disease.

Although milk is looked upon as an ideal liquid food, still it does not act as such in many cases, and, when given unmodified, may pass through the bowels in firm curds. The stool should, therefore, always be inspected when administering milk, as such curds may induce most unfavorable symptoms. A notable ill-effect of milk observed in some patients is tympanitis; this promptly disappears when the milk is discontinued. In young children it is always best to dilute the milk with barley-water or boil it and add sugar of milk or a maltose preparation.

Strained vegetable soup, is a most valuable food, and an agreeable change for the patient. Cream may be added to the soup. In the absence of diarrhea grape juice is permissible.

It is always best to increase the caloric food value of the milk by adding to it sugar of milk, Dextrimaltose, or Mellin's Food. In case of diarrhea the milk should be boiled. Ice cream, junket, gelatin and cup custard may usually be given during the febrile period. The best results are obtained by selecting the food best adapted to the case, and administering six to eight ounces every three hours. Some variation in the character of the diet is most agreeable to the patient and a great aid in keeping up the nutrition. The patient should also receive water freely.

The child must be sponged daily with cold or tepid water and when the fever runs high, remaining above 103 deg. F., during the greater period of the twenty-four hours, these baths may be repeated every three hours. After the bath the body should be vigorously rubbed, especially the extremities, until a good reaction sets in. An alcohol rub after the sponge bath usually induces a good reaction. Rubbing the body briskly with pieces of ice wrapped in a towel will have a most grateful and beneficial action in cases of hyperpyrexia. Should the patient react poorly after any form of cold water treatment it is better to desist and use milder measures, such as the tepid sponge bath or the pack, beginning with luke-warm water and gradually reducing the temperature of the pack to 85 deg. F. A Turkish towel wrung out of water of the proper temperature and wrapped around the body of the child makes an excellent pack.

In cases that are profoundly toxic I have employed the Murphy drip with good results. An enema should first be given and then the normal salt solution permitted to flow into the rectum at the rate of sixty drops per minute for two to three hours, and repeated after a two hour interval. Dextrose may be added in amounts of from five to six per cent.

Stimulation may become necessary in the later stages of the fever. The first and most prominent indication is cardiac weakness. Daily auscultation of the heart should be practiced and when the first sound loses its muscular element and resembles the second sound (embryocardia), small doses of whiskey may be given. Collapse will call for hot coffee, either by mouth or by rectum or camphorated oil subcutaneously.

Hemorrhage, if slight, requires nothing more than temporary withdrawal of food followed by greater caution in feeding, absolute quiet of the patient and possibly a change of remedy. When severe, it proves a grave complication. A cold application to the abdomen in the form of Leiter's tubes or an ice bag will prove of great benefit. Absolute rest must be enjoined, even the bed pan may be put aside and clothes used to collect the excreta. A blood transfusion is necessary if the loss of blood has been great.

Perforation and peritonitis are extremely fatal complications, although early laparotomy in perforation before peritonitis has set in offers better hope for the patient than conservatism according to the observations of Finney and Keen.

The leading typhoid *remedies* are *baptisia*, *bryonia*, *gelsemium* and *rhus tox*. The selected remedy should be continued throughout the entire course of the disease unless positive indications for a change occur.

The following indications embrace the most important symptoms of the leading remedies at our command.

Agaricus.—In typhoid fever where the nervous symptoms predominate. Low fever, tremulous tongue, and general tremor of the entire body. Among adults it is recommended for drunkards in whom the heart is giving out. Alcoholic stimulants must, of course, not be withheld from such cases. We often encounter boys who smoke cigarettes excessively and whose nervous system is about as wretched as the adult drunkard's. Here *agaricus* is well indicated.

Arnica.—General stupefaction of the senses; general sore-

ness, bed feels too hard; the sleep is disturbed by anxious dreams; the tongue is red and dry, with a brown streak down the centre; putrid taste in mouth; fœtor ex ore; involuntary discharge of feces and urine; the extremities become cold while the head remains hot; hemorrhages and bedsores develop.

Arsenicum.—Low types of typhoid, usually the later stages in unfavorable cases. Farrington cautions against the early use of *arsenic* in typhoid fever, and considers it a remedy capable of doing harm unless clearly indicated. It is most useful in the young or aged, or in those debilitated by previous ailments. The general symptoms so characteristic of *arsenic*, such as great restlessness, prostration; thirst for small quantities of water; hot, dry skin; general aggravation of all symptoms soon after midnight or noon; cadaverous smell of the discharges as well as of the patient, are all prominent indications for its use.

Baptisia.—The well-known mental symptom, the hallucination that the body is dismembered, that certain parts of the body are double, or that there is a second self in the bed with the patient, is a strong indication for *baptisia*, although its absence by no means deprives this drug of its usefulness in typhoid fever. *Phosphorus* and *petroleum* both have similar symptoms. The condition calling for *baptisia* is characterized by great weariness and a bruised feeling of all the limbs, together with a low type of fever and physical prostration; offensive diarrhea; breath, sweat and urine are alike offensive; there is dull stupefying headache; the patient is delirious, sleeps heavily and is aroused with difficulty. The tongue is dry and brown, the conjunctivæ are injected; the face is flushed and presents a besotted expression; exhaustion is marked. *Baptisia* may be indicated early in the disease when the symptoms are intense from the beginning.

Bryonia.—*Bryonia* may be indicated at any stage, although its most frequent application will occur during the first stage. The symptoms calling for its selection are very characteristic

and prominent—irritability, lassitude, desire to remain quiet and sleep; headache, worse from opening the eyes or moving the head; dryness of the lips, mouth and throat, with thirst for large quantities of water; aching of the limbs, worse from motion; frequent brown, putrid stools; delirium at night and restless sleep, disturbed by dreams of daily affairs; wants to go home; visions when closing the eyes.

Carbo veg.—*Carbo vegetabilis* is indicated in extreme cases. Many writers speak very highly of this remedy, but personally I am not able to say what *carbo vegetabilis* will do, as in such a condition I feel called upon to resort to stimulation and other adjuvant measures.

Gelsemium.—In the early stages *gelsemium* is frequently indicated on the symptoms of lassitude, drowsiness, dull headache, with heaviness of the eyelids and photophobia; slow, intermitting pulse, accelerated from slight exertion; chilliness up and down the spine; epistaxis; catarrhal condition of the eyes and respiratory tract; diarrhea.

Hamamelis.—Hemorrhages of dark, fluid blood from the bowels, with great soreness of the abdomen.

Hyoscyamus.—The delirium indicating *hyoscyamus* is characterized by loquacity, obscene actions, or even attempts at violence. The patient picks at the bed-clothes and grasps at flocks in the air, with continual muttering. *Stramonium* is similar, but the loquacity is confined to one subject and the patient is more noisy, often crying out in terror from supposed visions of horrible animals, bugs, and the like, which he sees coming out of the floor, crawling along the ceiling, etc. *Hyoscyamus* also has a total loss of consciousness, with dry tongue, involuntary stools, subsultus tendinum, dribbling of urine. I have seen small doses of *hyoscine hydrobromate* (1-1000 gr.) quiet a delirious patient after opium preparations had failed to exert any influence upon the condition.

Lachesis.—The *lachesis* patient, similar to the condition noted under *hyoscyamus*, is also loquacious, but he jumps from

one subject to another in an incoherent manner; there is stupor, dropping of the lower jaw; dry, red, or blackish tongue which is red at the tip and bleeding, and trembles on being protruded; the stools are horribly offensive, the abdomen sensitive to touch, and all symptoms are more intense after sleep. Purpuric spots on various parts of the body.

Mercurius.—The characteristic nocturnal aggravation, the greenish-yellow stools; broad flabby tongue and drowsiness may indicate *mercurius*.

Muriatic acid.—Low types of typhoid fever, in which the patient is stupid, sliding down to the foot of the bed; the tongue is parched and dry, difficult to protrude; stools involuntary while passing urine; loud moaning during sleep, and when awake not fully conscious of his surroundings.

Opium.—Either complete loss of consciousness with loud, stertorous breathing, contracted pupils, face dark red and bloated or pale with death-like expression, drooping of the lower jaw, hot sweat, or delirium with sleeplessness due to hyperesthesia of the special senses, so that slight noises keep him awake.

Phosphoric acid.—Low typhoid state, in which the patient becomes totally indifferent to his surroundings. He can be aroused, but with difficulty, and soon relapses into his apathetic condition. There is great debility, rattling of mucus in the chest, rumbling in the abdomen, tympanitis, grayish watery stools, bleeding from the nose, red streak down centre of tongue, milky urine, clammy skin.

Rhus tox.—After *bryonia* and *gelsemium*, *rhus toxicodendron* frequently follows. The provings of *rhus tox*. present a typical typhoid state, and the anatomical changes in the intestines closely correspond to the lesions of typhoid fever. The symptoms are sharp and well-defined, as is the case with *bryonia*. The mind becomes beclouded and the mental operations are performed with difficulty. The patient is restless from a distressing aching in every limb, and constantly changes

his position to gain relief (not as in *arnica*, where there is soreness produced by lying in one particular attitude, which makes him seek a new position). The sleep is restless, disturbed by dreams of great physical exertion. The lips are brown and dry, and the teeth are covered with sordes; the tongue is likewise brown and dry, presenting a triangular red tip. The diarrhea is worse during the night, often involuntary during sleep.

VARIOLA; VARIOLOID.

Variola, or *small-pox*, is an acute infectious, highly contagious disease, characterized by fever of a typical course, vomiting, intense lumbar pains, and an eruption of papules passing through the stages of vesicles, pustules and crust formation, the vesicles being umbilicated.

The nature of the *contagion* has not been determined. It is contained in the secretions, excretions and exhalations of the body, being especially disseminated by means of the dried scales and contents of the pustule. Pfeifer and others have constantly found small, homogeneous bodies in the epithelial cells surrounding the lesions. One or two are usually found in the cell substance. They probably belong to the class of protozoa (Park).

It attacks all ages, from the fetus *in utero* to the aged. A case came under my notice in which the eruption appeared in a new-born infant on the fifth day. During the last three weeks of her pregnancy, the mother had had an attack of varioloid, which was overlooked at the time on account of its mild nature. The infant died on the twelfth day. Among children it proves especially fatal. One attack protects against another, at least for a long period of time. The period of incubation is from nine days to two weeks.

The pock first consists of an area of round-cell infiltration into the *rete mucosum* in which a central area of coagulation-necrosis takes place. Inflammatory reaction occurs around this area, which represents the central depression of the vesicle,

with the formation of a reticulated vesicle containing serum, leucocytes and fibrin filaments. Pustule-formation supervenes, the leucocytes and cells of the rete mucosum becoming necrotic.

Symptoms.—The invasion is marked by a severe chill or repeated chills, with rapidly rising temperature. In children, convulsions are common at this period. Vomiting and intense backache are accompanying symptoms. In some epidemics the initial stage is marked by an erythematous eruption, either diffuse or measly, or by hemorrhagic exanthem which consists of extremely small punctate, closely aggregated pin-head sized hemorrhages into the epidermis. The temperature rises on the first day to 103° to 104° F., continuing with slight morning remissions until the evening of the third day when it reaches its highest point. On the fourth day it falls several degrees, this remission lasting until the seventh or eighth day, when there is a secondary rise—the suppurative fever.

The stage of eruption commences on the evening of the third day. Little red spots first in the face. If very numerous they coalesce, like measles-spots, with which they might be confounded if it were not for the granulated, shotty feel which they present to the sense of touch.

The eruption rapidly spreads to other portions of the body, and on the third day the papule is converted into a clear vesicle presenting an umbilication at its summit. The vesicle is also loculated. In the course of a few days (eighth day of the disease) the vesicle is transformed into a pustule, which dries up after a few days or breaks down, with the formation of a soft, yellow crust, later becoming brownish and dropping off, leaving a somewhat elevated spot which in time disappears. This occurs where the lesions are discrete and where the process has not extended into the deeper layers of the skin. Where, however, the deeper structures have been involved they adhere for a long time, leaving an uneven scar, which at first looks pink, but by degrees grows conspicuously white, producing the characteristic pock marks which are permanent.

Simultaneously with the appearance of the eruption upon the skin, identical lesions develop upon the mucous membranes exposed to the external air. Here it may result in great destruction of tissue.

Small-pox may run its course as a discrete, confluent, hemorrhage, gangrenous or malignant variety. The modified variety occurring in those partially protected by vaccination, and running a mild course without secondary fever, is described as *varioid*. In every other respect it is identical with true small-pox.

The *prognosis*, excepting in varioid, is always grave. As complications may be mentioned bronchopneumonia, pleurisy, septicemia, ulcerating keratitis, suppurating otitis, arthritis.

The *diagnosis* is often rendered difficult by the primary erythematous eruption. The true eruption may be confounded with measles in its early stages, but the sensation of balls of shot under the skin imparted to the finger by the papules of small-pox is a pathognomonic distinction, beside the severe initial symptoms of the attack. Again, in measles the temperature rises to its acme with the appearance of the rash, while in small-pox there is a temporary drop in the fever as the rash comes out.

From *varicella* it is distinguished by the intensity of its symptoms. Moreover, the eruption appears later than in varicella, does not come out in crops, is distinctly umbilicated, and presents a well defined inflammatory areola. The eruption of small-pox is also decidedly harder and more palpable than that of varicella.

Treatment.—As small-pox is one of the most serious and most dreaded of all contagious diseases, every precaution to prevent a spread of the same must at once be instituted when we are confronted by a suspicious case. The most rigid isolation and disinfection, as described under *scarlet fever*, must be carried out to the letter. Besides this every person in the house not recently successfully vaccinated (within four years)

should immediately undergo the operation. The patient must have as much fresh air as possible. If the fever is very high sponge-baths are indicated. Osler (*Practice of Medicine*) has come to the conclusion that the prevention of pitting is really not within the power of the physician. Protecting the ripening papules from light and keeping the hands and face covered with lint soaked in cold water or mild antiseptic lotions, is, however, to be recommended. The *red-light* treatment exerts no influence over pustulation (Schamberg). In the later stages we should aim to prevent the crusts from becoming hard and dry by the free application of vaseline. The addition of a little carbolic acid or boric acid to the vaseline is a distinct advantage.

In the early stages, *aconite*, *bell.*, *bry.*, *gelsemium* and *rhus tox.* are to be recommended. Jahr (*Therapeutische Leitfaden*) began all cases with *variolinum* as soon as the diagnosis could be established; and if, in spite of this remedy, the course became a grave one he followed with *sulphur*. He preferred these two remedies to all others.

Vaccinium is spoken of favorably by Goodno and others. From a limited personal experience with small-pox I have come to look upon *bryonia* followed by *rhus tox.* as the treatment most likely to exert a favorable influence over the disease. In the stage of suppuration when toxemia sets in *cinchona* tincture and whiskey should be freely used. When collapse threatens it may become necessary to resort to *strychnia*.

VACCINIA.

Vaccinia, or *cow-pox*, is an eruptive disease of the cow, the virus of which produces a lesion resembling the pustule of small-pox when inoculated into a human being. A specific organism has not been isolated from the vaccinia pustules nor is the true nature of the disease understood, some considering it a primary disease of the cow, while others believe it to be small-pox modified by its passage through animals. It has been experimentally demonstrated that children vaccinated with

cow-pox were not susceptible to inoculation with small-pox virus, the reverse condition also holding true. Pfeiffer and others have found small homogeneous bodies in the epithelial cells surrounding the lesions of both small-pox and vaccinia, and as small-pox virus has produced in cattle a disease indistinguishable from cow-pox, there is hardly any doubt that the two are due to the same micro-organism, modified by its transmission through the cow (Park).

A successful inoculation with vaccinia affords protection against small-pox in the majority of cases, at least for a number of years. Small-pox occurring in those who have been vaccinated usually assumes a mild course, *i.e.*, varioloid. As to the modifying influence of vaccinia upon small-pox already in progress there is a difference of opinion. According to Marson, if a person exposed to small-pox be vaccinated within four days, small-pox will be prevented; if later, but early enough to allow the vesicles to reach the stage of areola, the attack of small-pox will be modified; but later than this it is useless. Curschmann opposes this view as erroneous. It is interesting to know the views expressed by Hahnemann on this subject, which are no doubt borne out by the most trustworthy clinical testimony—"It is well known that when variola is added to cow-pox, the former, by virtue of its superior intensity as well as its great similitude, will at once extinguish the latter homeopathically and arrest its development. Cow-pox, on the other hand, having nearly attained its period of perfection, will, by its similitude, lessen to a great degree the virulence and danger of a subsequent eruption of small-pox, for which we have the testimony of Mühry and many others" (*Organon*).

The operation of vaccination consists of the introduction of the lymph from the vaccine vesicle of heifers into the circulation by bringing it in contact with a scarified surface for a sufficient length of time to permit of its absorption. Having cleansed the site of inoculation (usually the left arm, just below

the insertion of the deltoid muscle) with soap and water, followed by scrubbing with alcohol or ether, a few parallel scratches about half an inch in length are made with a sterilized needle, just deep enough to break the epidermis and expose the rete mucosum. A drop of *glycerinated vaccine lymph*, this being the most reliable and aseptic form in which the virus can be obtained, is placed upon the scarified surface and rubbed in gently with the needle. Guest (*Pediatrics*, Vol. IX, No. 5) has arrived at the conclusion that the entire contents of a tube is too large a quantity of lymph for the average child, judging from the results obtained in four hundred cases vaccinated by this method, in which there was more pronounced inflammatory reaction and more glandular swelling, besides the formation of a larger scab than in his former cases inoculated with points. Personally I have found that a single scratch, about one quarter of an inch in length and just deep enough to expose the rete mucosum is sufficient for a successful vaccination and lessens the danger of a severe "take" and of infection. After the lymph has been rubbed into the scarification a period of ten minutes should be permitted to elapse before applying a dressing.

Symptoms.—During the first three days after the operation, nothing excepting a slight local irritation, soon subsiding, will be noticed. On the third day, however, a papule appears at the site of inoculation, surrounded by an areola; this papule is converted into an umbilicated vesicle on the fifth or sixth day. The vesicle attains its maximum development by the eighth day, after which it becomes pustular. The areola gradually increases in size and depth of color until this time, but disappears as the acute symptoms subside. The pustule then dries up, forming a scab. On the twenty-first day the scab comes off, leaving the characteristic deep, pitted scar.

The constitutional symptoms accompanying vaccinia are fever, malaise, anorexia, etc., which begin with the formation of the vesicle, and attain their height at the period of pustula-

tion, after which they rapidly disappear. Swelling of the axillary glands is usually present.

Variations from the above-described course frequently occur. The vesicle may be late in developing or it may be premature and not fully developed in individuals who have been previously vaccinated. A generalized pustular eruption may accompany the primary lesion, which may persist in recurring attacks after healing of the same; or complications, notably erysipelas, ulceration and sloughing, glandular abscesses and *septicemia*, may develop as the result of faulty technique. Vaccinia may also occur as a general eruption of papules, which turn into vesicles and pustules. They appear on the face and extremities about the fifth day. I have also encountered a general papular rash occurring on the tenth day, looking like measles or the early stage of small-pox.

Deaths have occurred, but they were almost invariably from avoidable causes, as Voigt shows in his statistics. There is always a risk, however, in vaccinating a delicate, sickly child, and the operation should never be performed when an acute disturbance is present, or if there is a case of contagious disease in the family to which the child has been exposed. I have observed some anti-vaccinationists vaccinate, and their careless method has convinced me that they had good cause to be dissatisfied with their practice.

Besides, the invaccination of syphilis (when humanized virus was used) has occurred, and claims have been made that tuberculosis was likewise transmitted. This, however, has not been proved.

The age at which children are vaccinated is usually the third month, in the absence of any acute or constitutional illness. In the absence of an epidemic of small-pox I do not see the necessity for so prompt a procedure, especially if the infant is not in a good condition or is suffering with eczema. The advantages of early vaccination are that the general symptoms are usually milder and there is less danger of a secondary infection.

Some physicians, believing in the efficacy of vaccination to control whooping-cough, keep it in reserve to be employed as the opportunity manifests itself. All children, however, should be vaccinated before they are sent to kindergarten or school, and revaccinated at the period of puberty, or on the occurrence of an epidemic of small-pox.

Treatment.—The vaccinia lesion should be cleansed daily with alcohol, dusted over with boric acid powder and protected by a gauze dressing instead of a shield. When general symptoms occur one of the following remedies should be administered. *Belladonna* is indicated if fever, headache, diffuse redness and swelling about the site of eruption and glandular swelling develop. *Apis* or *rhus* may be indicated by erysipelatous manifestations. If there is much soreness with purulent secretion *hepar sulph.* should be given, or if the scab separates with suppuration and an unhealed ulcer remains, *silica*. I firmly believe that when vaccination is carried out on strictly aseptic lines, and the child is watched throughout as in the case of any other illness—being put to bed if necessary, and carefully prescribed for—none of the many complications and so-called constitutional after-effects, attributed to vaccination, will follow.

VARICELLA.

Varicella, or *chicken-pox* is an acute infectious disease characterized by the eruption of delicate discrete vesicles, which appear in crops, and disappear, in the course of a few days, by desiccation.

The specific virus has not been isolated, but it is known to exist in the vesicles, and can be transmitted by inoculation. The usual manner of contracting the disease is through contact with a case, although a third person may carry the infection. One attack protects against another. It may occur sporadically or epidemically. The period of incubation is usually two weeks.

The *symptoms* are slight in the majority of cases, but occasionally a severe type is encountered with extensive eruption, high fever and corresponding constitutional symptoms. In rare instances such complications as bronchopneumonia, nephritis and meningitis may occur. The chief clinical interest however attached to varicella is its superficial resemblance to small-pox and especially to varioloid.

The onset is abrupt, as a rule, the first signs of the disease being the appearance of papules and vesicles upon the trunk and extremities, accompanied by slight fever, anorexia, coated tongue and languor. Constitutional symptoms may be so slight as to attract no attention. Each day a new crop of vesicles makes its appearance; this usually continues for three or four days.

The eruption appears first as a small, red, slightly papular spot which is soon transformed into a clear, pearl-like vesicle. The vesicles are unilocular, although at times they give the appearance of being multilocular when they involve a hair follicle or sweat gland. They are surrounded by a faint areola, and do not become pustular unless infected by scratching, etc. In the course of a few days they dry up, the crusts soon falling off without leaving a scar, although in some cases a circular, pale area is left, which persists for some time, or, if ulceration has taken place as a result of infection quite a conspicuous scar may remain.

Varicella gangrenosa is a type of varicella which is attended by gangrenous stomatitis, as a result of infection in poorly-nourished or tuberculous children. If the process becomes extensive, it may prove fatal. As complications—which, however, are fortunately rare—may be mentioned erysipelas, adenitis, cellulitis, gangrenous dermatitis and nephritis. A *hemorrhagic type* of varicella is also occasionally seen in which purpuric spots about a quarter of an inch in diameter may be the initial lesion. The discoloration persists for a long time after the vesicles have dried up. It is not uncommon to have varicella

and one of the other infectious fevers occur simultaneously, although the error must not be made of considering those cases of varicella beginning with an erythematous or measles-like rash as cases of varicella plus scarlet fever or measles.

Diagnosis.—Varicella is to be differentiated from *small-pox* by the slight constitutional disturbances accompanying the rash, which appears abruptly, coming out in crops, and soon disappears by dessication, without pustulation or scar-formation. The eruption of small-pox may not always come out at once, and frequently new papules and vesicles will continue to appear for several days after the first lesions were seen. They do not, however, erupt in distinct crops, nor do we find lesions in the various stages of development, that is, fresh papules and vesicles interspersed among pustules, as is to be observed in varicella. Again, the papules of varicella lack the shot-like feel characteristic of the small-pox lesion, and the vesicles are more delicate and present a characteristic pearl-like appearance. There is no secondary fever in varicella. If the vesicle has not dried up by the fourth day, it is more likely small-pox or varioloid than varicella. The presence of a vaccination scar of recent origin is also presumptive evidence against small-pox.

Treatment.—In the presence of fever, rest in bed, a light diet, and, when there is much itching, the use of a dusting-powder, or olive oil and boric acid, is about all that is required in mild cases. *Aconite* may be called for in the beginning, to be followed by *rhus tox*. The gangrenous or pustular variety will call for *arsenicum*, *mercurius*, *rhus tox.*, etc.

PERTUSSIS.

Pertussis, or *whooping-cough*, is an acute infectious disease characterized by the presence of a paroxysmal suffocating cough and an associated catarrhal inflammation of the upper respiratory tract. It occurs both epidemically and sporadically, infection taking place through contact; seldom through the agency of a third person. The virus is disseminated by droplet infec-

tion during a coughing paroxysm or it may be conveyed by the sputum of an infected child. The specific bacilli disappear from the sputum after the third week and isolation of the case is unnecessary after that time.

Pertussis usually occurs epidemically in the spring and summer months although individual cases may be seen at any time of year. In recent years it has apparently become milder and more endemic in character. About 80 per cent of all cases occur in children under five years of age. A natural immunity is observed in about 30 per cent of the children exposed to the disease. The period of incubation is from three to eight days. One attack confers lasting immunity in most individuals.

Etiology.—Pertussis is caused by the *bacillus pertussis* of Bordet and Gengou. This organism may be isolated from the sputum collected at the end of a coughing attack by inoculating a plate of blood-agar medium with the same and incubating for forty-eight hours. The organism grows in minute, discrete, elevated colonies surrounded by an area of lightened blood. Morphologically it is a small bacillus resembling the influenza bacillus.

The *pathological processes* accompanying whooping-cough are catarrhal inflammation of the larynx, particularly in the region of the interarytenoid cartilages; tracheitis and more or less bronchitis; swelling of the bronchial glands; rhinitis. In fatal cases bronchopneumonia with emphysema and areas of atelectasis are the most common lesions found; there may also be entero-colitis and cerebral congestion, with effusion and cortical hemorrhages. The toxin of whooping-cough in some cases appears to affect the smaller blood-vessels and favor hemorrhagic extravasations, either spontaneous or as a result of the congestion which is associated with the cough-paroxysm. Moebius believes that the nervous system may also be acted upon by this toxin in a manner somewhat similar to the action of the diphtheria toxin.

Symptoms.—The course of whooping-cough is in three

stages: the premonitory, or catarrhal; the paroxysmal stage, and the stage of decline. The first stage usually lasts one to two weeks; the second stage may persist for a month, while the stage of decline is a gradual lessening of the number and severity of the coughing spells and persists as long as there is any bronchitis present. The duration, therefore, depends to some extent upon the child's general condition, the severity of the attack and the presence of complications. Even after apparent recovery, a fresh cold may bring about a recurrence of the cough and whooping spells. The average duration of an ordinary case is about six weeks, but the course is influenced by treatment and by the child's recuperative powers and the occurrence of a complication.

The attack begins as an ordinary cold, indistinguishable in the beginning from a simple upper respiratory infection, with, however, this difference, that instead of yielding to treatment in the course of a few days, or abating of its own accord, the cough gradually increases in frequency and severity, soon assuming the paroxysmal and spasmodic type characteristic of the disease. An early symptom that should always arouse suspicion is the nocturnal aggravation of the cough from the very beginning. Another diagnostic sign of value at this stage of the disease is the presence of a lymphocytosis.

Examination of the chest at this time reveals nothing beyond a slight bronchitis. In the very beginning there is usually indisposition, running of the nose, a short, dry cough, and slight fever. These symptoms soon abate, but the cough increases in severity. The cough is characterized by a sudden, loud expulsive effort, followed in rapid succession by similar efforts of gradually decreasing force; through these continued explosions the chest is almost completely emptied of air, so that the child is obliged to draw in a deep breath at the end of the paroxysm. As the glottis is narrowed during this long-drawn inspiration, a loud, piping sound is produced, constituting the whoop, from which the disease is named. As soon as the lungs have been re-

filled the cough begins anew, consisting, as before, of rapidly following expulsive efforts, ending with the whoop. This continues (two to six coughing fits) until the paroxysm is terminated either by the dislodgement of a plug of mucus from the trachea, or by the vomiting of the ingesta or of a quantity of tenacious mucus.

During such an attack the face becomes red, even livid; the eyes are injected and bulging, and the child clings to the nearest object for support, or stands with the feet wide apart and the hands resting upon the knees. Bleeding from the nose frequently occurs during the paroxysm, and cortical hemorrhages from the meningeal vessels may occur in severe cases. When such a hemorrhage is extensive, hemiplegia and convulsions will follow. This hemorrhagic tendency is one of the most serious aspects of whooping-cough. Sub-conjunctival hemorrhage is quite common, as are also minute hemorrhages about the eyes and face. No doubt the action of the pertussis toxin upon the blood-vessels is responsible for the condition.

The number of paroxysms in a day will vary greatly in different cases. They are usually more frequent during the night. In very young children the cough is not as characteristic as in older ones, the whoop being especially faint or indistinct, but the same paroxysmal nature of the cough is present and cyanosis is more pronounced.

The face gradually assumes a bloated appearance from the recurring vascular engorgement, and the eyes are deeply injected; slight hemorrhages may be seen under the conjunctiva. The eyes are unnaturally moist. Under the tongue a characteristic sign is frequently seen, namely, ulceration of the frenum. This is induced by the repeated propulsion of the tongue over the lower incisor teeth in coughing. In my experience it has only been present when there was at the same time catarrhal stomatitis in association with the whooping-cough, rendering the mucous membrane particularly vulnerable.

With the decline of the disease the paroxysms become less

frequent and less severe, soon losing the spasmodic character of the cough, and the expectoration becomes muco-purulent, as in an ordinary bronchitis. With a fresh cold the whoop may reappear ("after-pertussis"). This does not indicate a relapse in the sense of a reinfection but simply the persistence of the spasmodic habit which has been established and every cough will show evidence of this element for a long time after an attack of pertussis. With the advent of pneumonia, however, the cough loses its spasmodic character.

The commonest *complications* of whooping-cough are *bronchopneumonia* (in the winter months) and *entero-colitis* (summer months). The advent of bronchopneumonia is recognized by the appearance of fever, together with rapid respirations and dyspnea, and subcrepitant râles throughout the chest. The cough loses its paroxysmal character during the height of such a complication.

Diarrhea is liable to become a troublesome symptom in delicate children, especially during the summer months.

Convulsions are frequent among infants. They may be due to asphyxia, meningeal hemorrhage or pneumonia. *Meningitis* rarely, if ever, results from whooping-cough, although marked meningeal symptoms due to hyperemia of the brain and edema of the pia mater may be observed.

Dilatation of the heart, due both to the strain on the heart as well as to the action of the toxin upon the myocardium, may be observed (Koplik).

As a *sequela*, tuberculosis is most to be dreaded. Whooping-cough, as is well known, is one of the most potent predisposing causes of tubercle, ranking second to measles in this respect. This is due to the fact that in both of these diseases bronchitis and enlargement of the bronchial glands is a constant occurrence.

The *prognosis* depends to a great extent upon the age and previous health of the child. Normal children above five years of age seldom suffer great inconvenience or serious after-

complaints under proper treatment. The prognosis becomes grave when bronchopneumonia is added, or where the hemorrhagic tendency is marked. In the case of infants the prognosis is more grave. There is a greater tendency to pneumonia at this age and the infant is less able to withstand the exhausting coughing spells and the constant vomiting.

Diagnosis.—During the prevalence of an epidemic the diagnosis should present no difficulties. Isolated cases, however, may become puzzling, especially when atypical. The character of the cough, together with the accompanying signs described under the symptomatology, should help out in differentiating whooping-cough from an ordinary bronchitis.

Prof. Filatow, of Moscow, confirms the researches of Hippus and Blumenthal, who noticed that pertussis patients have a *pale urine of high specific gravity*. The occurrence of a *lymphocytosis* during the catarrhal stage is also suggestive of pertussis.

Hyperplasia of the *bronchial glands* frequently provokes a paroxysmal cough, but the course is a chronic one, and there is associated bronchitis, and usually tuberculous foci elsewhere in the chest. Other possibilities of error are found in the so-called "*spasmodic bronchitis*" of infants, and *catarrhal laryngitis* (false croup).

Treatment.—Isolation is difficult to carry out, as the disease is already contagious during the stage at which it cannot always be recognized. Nevertheless, every effort should be made to protect delicate children and infants against exposure by excluding from their presence, during an epidemic, all children with colds or coughs. Prophylactic injections of pertussis vaccine have been used extensively in recent years. Hess in 1914 gave prophylactic vaccine to 244 children during an epidemic in an Infant Asylum and only 6 cases developed the disease. Shaw, of the New York State Board of Health, reports 164 children exposed to pertussis who received vaccine and only 11 cases contracted it. He advises the use of large doses, namely 500 million for the first injection, one billion for the

second and two billion for the third, giving the injections every second or third day.

The patient should receive as much air as possible, and in pleasant weather may be permitted to be out-of-doors. Protracted cases do well from a change of climate, the seashore being particularly beneficial.

If the cough is very troublesome at night, and especially in the case of infants in whom asphyxia is to be feared, the vaporizing of *cresoline*, *creasote* or *oil of eucalyptus* in the sick room is often attended with good results. Holt prefers *creasote*, vaporized in a croup-kettle.

The *remedies* recommended for whooping-cough are legion, and space forbids enumeration of so long a list. While there are, perhaps, a dozen which are used a hundred times when the others are used but once, still it is impossible to tell just which remedy will be of the greatest benefit in a given case before the symptoms have been carefully considered. The popular feeling as to the clinical value of our remedies in this affection is well presented by the following statistical report by Dr. Geo. B. Peck (*Trans. American Institute of Homeopathy* 1898): "Out of every thousand prescriptions by members of this Society for the amelioration of that group of morbid phenomena popularly designated whooping-cough, at least 175 are for *drosera*, 153 for *belladonna*, 123 for *ippecacuanha*, 76 for *cuprum* (*metallicum* and *aceticum*), 54 for *corrallium rubrum*, 44 for *antimon. et pot. tartaricum*, 24 for *mephitis*." etc.

In the early stages *aconite*, *bell.*, or *ippecac* may be indicated. When the characteristic spasmodic cough is established *belladonna* is the chief remedy. This may be later alternated with *ippecac* as vomiting is a prominent symptom or *tartar emetic* when there is considerable bronchitis with rattling of mucus in the large tubes.

The following remedies should be studied for special symptoms and complications:

Arnica.—Painful paroxysms (*bryonia*); tendency to hemorrhages; meningeal hemorrhage.

Bell.—Intense redness of face during paroxysm; mental excitement; child becomes very much frightened from the coughing paroxysms and awakens with suffocating spells at night. Convulsions. The most important remedy in the early stage.

Carbo veg.—Protracted cases. Follows well after *drosera*. Hoarseness; anemia; sluggish circulation; flatulent indigestion.

Coccus cacti.—Cough, especially worse in the early morning, followed by the expectoration of yellowish or bloody, tough mucus. This remedy has proven of value during the early paroxysmal stage when abundant, stringy expectoration is present.

Drosera.—Paroxysmal stage. Worse after midnight; gagging and vomiting predominate; the expectoration is frequently blood-streaked; tuberculous diathesis. Personally, I have been disappointed in the results seen from this remedy but many homeopathic physicians consider it specific for pertussis.

Hyos.—Incessant cough when lying down, relieved by sitting up.

Ipecac.—Spasm of the glottis before paroxysm; the child stiffens out during the cough and becomes blue in the face. Bronchopneumonia, with abundant fine râles; vomiting after cough. The expectoration is often blood-streaked. Hughes recommends beginning all cases with *aconite* and *ippecac* in alternation.

Mephitis.—During the spell the child passes both urine and feces; diarrhea and flatus very offensive; the child must be taken up during the cough, turns blue in the face and seems asphyxiated. *Mephitis* is rarely used excepting in severe types of the disease.

Naphthalin.—Goodno recommends this remedy to be used as soon as the case is recognized. He employs the first decimal trituration.

Tartar emetic.—Bronchopneumonia. Rattling of mucus in larger tubes; deficient oxygenation of blood. As a routine prescription, used in alternation with *belladonna*, this is one of the most useful remedies.

Vaccine therapy has been tried in the treatment of pertussis but its chief value lies in its prophylactic effect. Three injections, of 500 million, should be given at three days intervals. After the disease has become fully established the results from the use of a vaccine are disappointing.

PAROTITIS.

Epidemic parotitis, or *mumps*, is an acute infectious disease in which the parotid glands become inflamed and markedly swollen. The specific contagion is not known, but it no doubt gains access into the gland through the duct of Steno, setting up an intense hyperemia, followed by a profuse serous exudation (soft swelling). The process begins in the ducts and acini of the gland, rarely extending to the interstitial connective tissue, and never terminates in suppuration. For this reason resolution is perfect in the vast majority of cases, as the tumefaction is the result simply of hyperemia and edema and not of structural changes in the gland.

Secondary parotitis is an infection of the parotid gland (usually one-sided), with pyogenic micro-organisms, occurring during the course of one of the infectious fevers. It may complicate typhoid fever, diphtheria, scarlet fever, small-pox and measles, rendering the prognosis grave. In these cases the submaxillary gland is rarely spared. It differs from mumps in terminating in suppuration, the entire parenchyma of the gland being more or less involved in the destructive process.

Mumps appears epidemically, although never to the extent attained by epidemics of the other prominent contagious diseases of childhood. Close contact seems necessary for infection. It is most prevalent during the damp seasons of the year. The period of incubation is from two to three weeks. One attack gives immunity against another.

Symptoms.—For a day or two there may be a slight fever with lassitude, restless sleep, nervous irritability, loss of appetite, etc., preceding the appearance of the characteristic lesion.

The inflammation of the gland induces first a painful stiffness of the jaw and tenderness in the region of the parotid. Swelling rapidly sets in, and in the course of a few days the gland will be swollen to its utmost extent. The fever may increase and the sleep become disturbed by restless dreams or delirium; convulsions have been known to occur in young children. The left parotid is the one most frequently attacked first. In the majority of cases the opposite side begins to swell in a day or two after the appearance of the first lesion. Sometimes the opposite parotid is not involved until the first begins to subside, or it may escape entirely.

At the height of the disease the face presents a ludicrous appearance. The entire parotid region stands out prominently from the presence of a tense, shining swelling which spreads anteriorly to the zygoma and posteriorly to the sternocleidomastoid. The tumor feels firm over its centre while the edges pit on pressure. The enlargement is uniform and regular, not nodular as in lymphadenitis. It is also perfectly immovable, for the parotid gland is so firmly held down by the deep fascia as to render its displacement impossible.

The fever now gradually subsides, usually not lasting more than from three to four days, but the patient is extremely uncomfortable, every effort at opening the mouth being attended with pain, and any article of food not bland in character frequently exciting intense suffering. In fact, the pain produced by taking anything acid into the mouth is looked upon as pathognomonic and a symptom of diagnostic value. The swelling attains its height within three or four days, subsiding by the end of a week.

Metastases to the testicle in the male and to the ovary or breast in the female are not uncommon in older children at this time, *i. e.* during the stage of decline, but in young children this does not occur. Aside from the possibility of such a complication the *prognosis* is good.

Secondary parotitis occurs during the course of one of the

acute infectious diseases, and begins as a hard, painful swelling, more circumscribed than in mumps, with an inflammatory blush soon showing itself over the surface. This gradually deepens in color; the swelling becomes more tense, and points of fluctuation can be elicited. The gland breaks down and pus can be expressed from its duct. The *prognosis* is always grave, although it is said to be less so when occurring late in the course of the disease which it complicates.

Diagnosis.—It seems unnecessary to call attention to the question of diagnosis in a simple case of mumps, yet errors are sometimes made. One of the most frequent is the mistaking of acutely enlarged *cervical lymphatic* glands for mumps; here the slower onset, the multilocular feel of the tumefaction and its movability will readily distinguish this condition from mumps. Furthermore in mumps the swelling begins in front of the lobe of the ear while in adenitis it is either below or posterior to the ear. *Diphtheria* with pronounced swelling of the cellular tissue of the neck, has likewise been mistaken for mumps, as has also mastoiditis. A sign to which attention has recently been called is redness and pointing of the orifice of Steno's duct on the affected side.

Treatment.—The most important remedy is *belladonna*. It corresponds to the vascular engorgement, the fever, and the associated symptoms.

Mercurius may be indicated early when there is but slight fever, pale swelling of the parotid region and gastric derangement. It is useful in the later stages of all cases to assist absorption of the inflammatory products.

For metastasis to the testicles *pulsatilla* and *clematis* are the chief remedies. If induration with tendency to atrophy follows, *aurum* should be considered.

Metastasis to the ovaries calls for *apis*, *cimicifuga*, *pulsatilla*, *hamamelis*.

Secondary parotitis finds in *rhus tox.* its most useful remedy. As the process advances, *hepar* or *arsenic* usually becomes indi-

cated. *Calc. sulph.* is the main remedy to promote healing after pus has been discharged either through fistulous openings or by means of an incision. As soon as the gland becomes swollen, hot fomentations wrung out of a 1 to 4,000 solution of the bichloride of mercury should be applied continuously.

INFLUENZA.

Influenza, or *la grippe*, is an acute infectious disease occurring pandemically and attacking all ages alike. It is characterized by fever of sudden onset and short duration, accompanied by marked prostration and complicated with either catarrhal inflammation of the respiratory or alimentary tract, or by certain nervous phenomena. These characteristics distinguish true influenza from the endemic cases of upper respiratory infection commonly designated as *grippe*.

The *bacillus of Pfeiffer* is the exciting cause, being found in almost pure culture in the sputum of influenza patients. It is a short, thin rod with rounded ends; it does not stain by Gram's method and is best demonstrated with dilute fuchsin. It is difficult to cultivate; besides it usually disappears from the sputum early and for this reason its presence is often missed.

The period of *incubation* is short, usually from one to three days. One attack does not afford immunity against another, as is the case in many of the epidemic infectious diseases; on the contrary, it may even lead to an increased susceptibility to a fresh attack, or, reinfection may set in from a focus in the nose and throat, ears or bronchi.

While influenza, as a rule, pursues a short and acute course, nevertheless it shows a tendency to become protracted in many instances, sometimes becoming latent for a time and then suddenly flaring up with acute manifestations. Again, bronchitis may persist for weeks, the secretion showing influenza bacilli in pure culture (Ortner, *Modern Clinical Medicine*, 1905) and a bronchopneumonia of a protracted course may likewise be due to the influenza bacillus, these cases presenting particular

difficulty in their differentiation from pulmonary tuberculosis (Wassermann).

Symptoms.—The disease begins abruptly with fever, severe headache, general aching and prostration. The fever remains at its height for a period of from three to five days, during the entire course of which prostration is marked, and headache and muscular aching are usually very distressing. A symptom present at this time and upon which Furbringer, of Berlin, lays great stress, is marked redness of the face. This shows itself as a diffuse flush and differs from scarlet fever in the absence of the white line about the mouth and pallor of the forehead. As Furbringer also points out, there is often present a slight icteric discoloration of the skin, although there is not much evidence of bile in the urine. During the epidemic of 1918 many cases ran a characteristic temperature curve in which there was a high fever of abrupt onset for three days which fell to or near normal and then again rose with the appearance of chest symptoms (bronchitis or bronchopneumonia). The symptoms noted were cough, increase in the fever, increased pulse and respiratory rate, and the development of dry and moist râles in the bronchi and subcrepitant râles at one or both bases of the lungs. In unfavorable cases cyanosis, increasing prostration and circulatory failure with a terminal pulmonary edema occurred.

Several clinical types are to be encountered, depending upon the predominance of catarrhal or nervous symptoms and the locality chiefly attacked. Thus, there is the *cerebral form* characterized by a predominance of headache, together with delirium, and even unconsciousness, some of these cases simulating meningitis; the *abdominal form*, characterized by vomiting, anorexia, gastralgia, diarrhea, some with predominance of gastric symptoms, others simulating typhoid fever; the *neuralgic form*, in which there are neuralgic pains in the peripheral nerves and other regions; the *thoracic form* complicated by bronchopneumonia, and the *catarrhal form*, the

commonest variety, in which catarrh of the upper respiratory tract is the most prominent symptom. Extreme prostration, however, is common to all forms, this being the chief feature of the disease. The toxin exerts a most potent influence upon the nervous system, which manifests itself as prostration, cardiac weakness and neuralgic pains, and during convalescence in the persisting prostration and the strong tendency to the development of neurasthenia, perineuritis, insomnia, persistent headache, and even insanity. Fortunately these complications are not as common in children as in adults, and, taken altogether, the prognosis is better, although a complicating bronchopneumonia is always of serious import. As in the case of measles and whooping-cough, a predisposition to infection with the tubercle bacillus is created.

Nephritis may occur in influenza; sometimes this is of the hemorrhagic type.

Rhinitis and *acute otitis media* are among the commonest complications. There is less tendency to mastoiditis than in scarlet fever but the otitis often runs a protracted course.

Bronchopneumonia is the most serious complication and is the cause of the majority of fatalities. *Empyema* is usually due to secondary infection with the streptococcus and is more serious than the form following lobar pneumonia.

The *prognosis* depends upon the age of the patient, the previous health and the presence of complications. Filatow lays stress upon the fact that in childhood it is mainly during the first to third year that the grave cases are encountered.

The *diagnosis* seldom presents difficulties during the prevalence of an epidemic, but isolated cases may be mistaken for a variety of other affections, particularly in the beginning. The catarrhal symptoms, hard cough and drowsiness may lead to a suspicion of beginning *measles*, but the subsequent course soon corrects this error. From *pneumonia* it is to be distinguished by the absence of physical signs limited to a pulmonary lobe; the presence of a leucopenia and the short course of the primary

fever. When pulmonary symptoms develop during the secondary rise in the temperature they are in the nature of a complication. Bacteriological examination of the sputum and nasal secretion may or may not give positive information. Cerebral cases may simulate *meningitis* or *cerebro-spinal meningitis*. The mild cases of *grippe* (upper respiratory infection) above alluded to present none of the profound toxic manifestations of influenza. In *protracted cases* the condition is often very puzzling. Such cases may simulate tuberculosis. Here the absence of the characteristic apical signs of tuberculosis and the bacteriological examination of the mucous secretions are the most conclusive diagnostic data.

Treatment.—The child should be kept in bed, absolute rest enforced, and strict isolation carried out.

The diet should be highly nutritious, but to prevent gastrointestinal complications, easily digested food only should be selected. When the pulse becomes weak and irregular a moderate amount of whisky should be administered at regular intervals. During convalescence a change of climate may be desirable and milk and eggs should be given in conjunction with the regular diet.

The most important remedies are *aconite*, *gelsemium* and *bryonia* in the beginning of the disease. *Aconite* is most useful in cases of sudden onset with high fever before any complications have developed. *Ferrum phos.* is useful for the early stage of the bronchitis, the symptoms upon which it is chiefly prescribed being a hoarse cough with blood-streaked expectoration. *Belladonna* is frequently indicated by the flushed face, somnolence and hard, barking cough. When the cough is deep and painful, *bryonia* is indicated, either alone or in alternation with *aconite*. For the pneumonic complications *phosphorus*, *tartar emetic* and *scilla maritima* are the most frequently useful remedies.

Arsenicum is indicated where the prostration is extreme and presents the chief manifestation of the disease. There may also

be sneezing; acrid, watery coryza; the process extending to the chest, with cough and dyspnea; great restlessness.

Bryonia.—Pains in the muscles, every limb aching intensely; lies perfectly quiet and does not wish to be disturbed; dry, painful cough. Bronchopneumonia complicating influenza (*ant tart., phosphorus*).

Eupatorium perf..—Deep-seated aching in the back and extremities, as if the bones would break; the skin is slightly jaundiced and the tongue heavily coated; bilious vomiting.

Gelsemium.—The symptomatology of *gelsemium* presents a true picture of the average case of *grippe*. The condition begins with lassitude and chilliness; “creeps” especially up and down the back, and the patient hugs the stove to get warm. He feels prostrated, every part of the body aches, and he complains of headache, soreness and sensitiveness of the eyes, obstruction of the nose, sore throat and prostration. The soft, weak pulse, heavy eyelids and flushed appearance of the face are very characteristic of *gelsemium*.

Pulsatilla.—Catarrhal symptoms predominate; mild, tearful disposition, the tongue is heavily coated and covered with viscid saliva, but there is no thirst; the patient is constantly chilly; otitis media.

MALARIA; MALARIAL FEVER.

Malaria represents a group of febrile affections resulting from infection with micro-organisms belonging to the class of protozoa. Each type of malarial fever is traceable to a distinct variety of micro-organism, possessing its own morphological and biological peculiarities. There is a specific parasite for tertian intermittent fever, for quartan intermittent fever, and for estivo-autumnal fever, or tropical malaria. These parasites attack the red blood-corpuscles, in which they live and develop to full maturity and sporulation. With the completion of sporulation a malarial paroxysm occurs. The tertian organism requires forty-eight hours to undergo a complete develop-

mental cycle; consequently a patient infected with this parasite will experience a paroxysm every third day, *i.e.*, with the occurrence of sporulation. Infection with the quartan parasite results in a paroxysm occurring every fourth day. Double infection with the tertian parasite, each group maturing on separate days, results in daily paroxysms. This is the most frequent type in the acute intermittent fevers in this latitude (Osler). Quartan fever is extremely rare in this country. This parasite may be present in the blood coincidentally with the tertian parasite. By such a combination most puzzling types of fever are produced. The parasite of estivo-autumnal fever is smaller than the other types of parasites, and is practically confined to the Southern States in this country.

The disease prevails endemically in certain localities, which are known as malarial regions. Although low, swampy and poorly drained regions and the banks of sluggish streams are the most frequent localities for malaria, still it also exists in many of the larger cities, especially in their suburbs and along the river fronts. The disease is conveyed to man by the sting of the mosquito, the genus *anopheles* being the one capable of acting as a host for this parasite. Malaria has no doubt increased in the northern cities since the influx of laborers from the South and from Italy has grown to such proportions.

The *pathological changes* resulting from malarial infection are extreme anemia, due to destruction of the red corpuscles by the parasite; enlargement of the spleen, which may lead to hyperplasia of the same; pigmentation in the liver, kidneys and brain. In cases which have resulted fatally there may be intense pulmonary congestion or pneumonia; nephritis; gastroenteritis. Fortunately, fatal cases are rare, the pernicious form of malarial fever being quite uncommon in this locality.

Symptoms.—A typical malarial paroxysm, consisting of three well defined stages, namely, chill, fever, and sweat, is seldom seen in young children. Both the first and third stages may be absent or poorly defined. Instead of a chill there may

be only the signs of a vasomotor spasm, such as blueness of the finger-nails, cyanosis of the face, cold extremities and yawning, or there may be vomiting, diarrhea and even convulsions or a comatose state preceding the accession of fever. In the course of an hour or less the fever rises rapidly and may reach an alarming height. This condition of hyperpyrexia lasts for an hour or two, ending by a gradual fall. Sweat may be entirely absent.

When there is a complete remission of fever the child may appear well until the second paroxysm occurs. As the attacks recur they become more and more atypical, and a remitting fever may develop.

Enlargement of the spleen and anemia develop, especially if the disease has lasted for some time. The symptoms accompanying the febrile stage are those common to febrile disturbances in general.

The *prognosis* is usually good. Untreated cases may take one of the following courses: (1) mild cases may go on to spontaneous recovery; (2) the paroxysm may gradually diminish in intensity, but grave anemia and chronic cachexia develop, or (3) the paroxysm may increase in severity and assume finally a pernicious type (Thayer, *Lectures on Malarial Fever*, 1897).

Masked or Irregular forms of Malaria and Malarial Cachexia. A malarial paroxysm may be so atypical, or affect a certain region to such a degree, as to entirely mask the condition, the malarial element only being eventually suspected by the regularity of recurrence of the attack, the association of enlarged spleen and anemia, and possibly by a history of exposure to malarial infection or residence in a malarial district.

Disturbances in the nervous system are common. Headache, continuous or recurring; neuralgia in various localities; intermittent spasmodic torticollis, accompanied by a slight rise in temperature and enlarged spleen (Holt); multiple neuritis. Trigeminal neuralgia is rare in children. Congestion of the lungs, simulating pneumonia, may occur paroxysmally.

Malarial cachexia may develop likewise without malaria having been suspected, either from the attacks being unaccompanied by very high fever, or from presenting themselves in a masked form. The child is anemic and emaciated, the skin being dry and sallow. The face has a drawn, pinched look, and the eyes are surrounded by dark circles. Indigestion and diarrhea, irregular febrile movements and enlargement of the spleen are usually present.

Diagnosis.—Malarial infection should always be suspected when a periodic disturbance, accompanied by anemia and enlargement of the spleen, is encountered. In order to remove all question of doubt, a blood examination should be made. A negative result does not necessarily exclude malaria, as it may require several examinations in order to find the plasmodium. Even in the absence of the plasmodium a leucopenia together with an increase in the large mononuclear leucocytes is suggestive of malarial infection.

Anemia infantum pseudo-leukemica presents some of the symptoms of malarial cachexia, but the absence of fever, the leucocytosis and absence of the malarial parasite readily differentiate the two conditions.

The remittent form of malarial fever is frequently confounded with such conditions as the *hectic fever of tuberculosis*, *typhoid fever*, and the *septic fever of empyema, pyelitis*, etc. A careful process of exclusion is therefore necessary in order to justify a diagnosis of malaria. Finally, the therapeutic test is of value and may be tried in doubtful cases.

Treatment.—Little can be done for the patient during a paroxysm. During the interval and during convalescence a tonic treatment is indicated. Cases simulating typhoid fever are to be managed on the same general principles applying to such cases.

Remedies prescribed in malarial fevers are usually divided into three classes: (a) those possessing a specific and abortive influence over the paroxysms, (b) those indicated for general

disturbances arising during and complicating the paroxysm, (c) those indicated in the chronic form and for the cachectic manifestations.

To the first class *cinchona* and its alkaloid, *quinine*, belong pre-eminently. We must all admit its definite action in typical cases of malarial fever and accept it as the specific remedy for the disease.

The true sphere of *cinchona* lies in that class of cases which presents each stage well marked, with the absence of any complications or symptoms not directly traceable to the febrile paroxysm. *Chininum sulph.* is supposed to exhibit greater regularity in the time of occurrence of the paroxysm, besides possessing some symptoms not found under *cinchona*. For a fuller description of these remedies and their special indications in intermittent fever I must refer to Allen's *Therapeutics of Intermittent Fever*. As to the dose, that is unfortunately a matter of contention. Kafka (*Homeopatische Therapie*) sums up his experience as follows: "Given on exact indications, *quinine* acts in small as well as in larger doses, but not in infinitesimal doses. While the most beautiful results were attained with the 1x trituration, or even stronger doses of one to two grains given every two hours during the period of apyrexia, we exerted ourselves in vain with the 2d, 3d, etc." Goodno (*Practice of Medicine*) expresses similar views and he recommends the usual therapeutic dose. Personally, I believe that it is necessary to give a sufficient amount of *quinine* to destroy the plasmodium in order to cure the case.

In malarial cachexia *arsenicum* is the most important remedy.

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